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PHYTOPATHOLOGY

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Official Organ of The American Phytopathological Society

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SPINDLING OR HAIR SPROUT OF POTATO

WILLIAM C. SNYDER, H. EARL THOMAS, AND S. J. FAIRCHILD

(Accepted for publication June 16, 1946)

When potato tubers (*Solanum tuberosum* L.) produce sprouts that are slenderer and weaker than normal, the disorder is generally referred to as spindling sprout or hair sprout, depending upon the severity of the symptom. Although various causes, especially a virus (9, 25), have been suggested during the past 30 or 40 years, the nature of much of the spindling sprout that occurs today remains obscure (12).

In 1942, an outbreak occurred in plantings of the White Rose variety of potato along the coast of California. The findings reported here on the nature of spindling sprout are a result of investigations initiated then. They apply, however, only to the disease as it has occurred along the California coast, since no study has been made of spindling-sprout tubers from other regions.

Economic loss from spindling sprout results from the failure of affected planting stock to give good stands, and the failure of plants from affected stock to produce satisfactory yields.

SYMPTOMS

Affected uncut tubers sprout prematurely, though seldom—in California—immediately after harvest. Most of the eyes of such tubers produce sprouts varying from slightly longer and more slender than normal to threadlike growths (Fig. 1) up to a foot or more in length. Tubers about the size of a pea or smaller may be formed at the tip or elsewhere on the spindly sprout. Severely affected tubers often fail to sprout. Diseased tubers are somewhat brittle and are low in starch and specific gravity. Similarly affected tubers in Minnesota are reported (11) to be high in reducing sugar and total sugar and low in dry weight. The differences are not sufficiently great, however, to permit separation of affected and healthy tubers with certainty before sprouting.

EVIDENCE AS TO THE CAUSE OF SPINDLING SPROUT

Spindling sprout has been attributed, at least tentatively, to hot, dry weather of the preceding growing season, Fusarium wilt infection, infection by the leaf roll, witches' broom, aster yellows or some other virus, faulty storage conditions, purple top, or some other less tangible cause (2, 3, 5, 6, 8, 13, 14, 15, 16, 17, 18, 19, 21, 28, 29).

Several workers (11, 17, 20) have used grafting as a means to determine whether spindling sprout is caused by a virus. The results have been, on the whole, adverse to such a conclusion.

However, in order to test further the hypothesis that the spindling sprout

in California might be caused by a virus, several attempts were made to transmit the disease by grafting. Sprouted eyes from healthy tubers were replaced with eyes from sprouted diseased tubers, and vice versa. Also, spindling sprouts on diseased tubers were inarched to normal sprouts on healthy tubers. In none of these experiments did any change develop in the grafted parts or in the sprouts from undisturbed eyes of tubers.

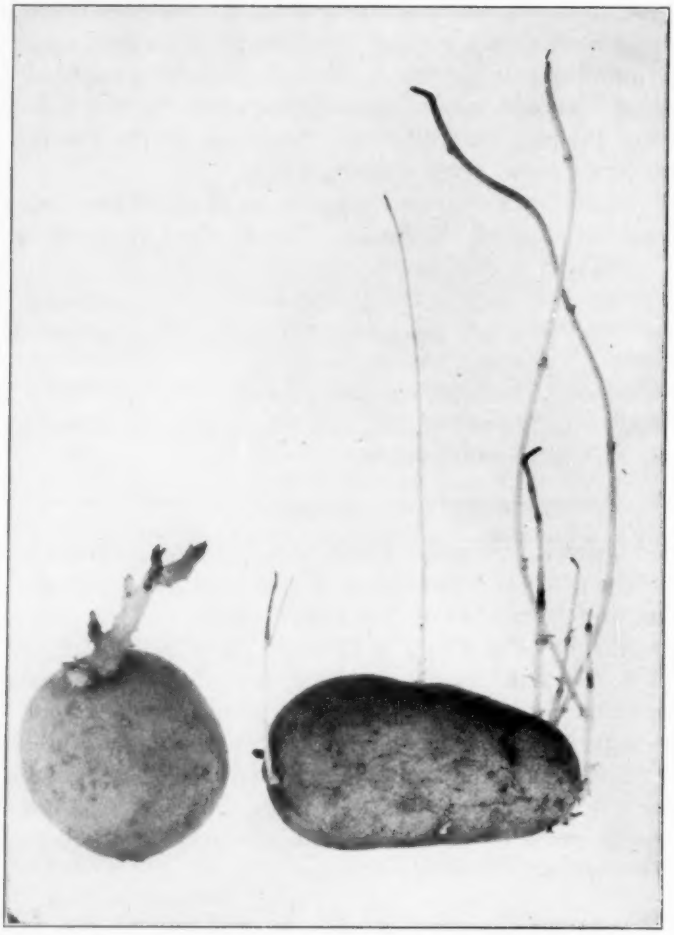

FIG. 1. Sprouted tubers from plant not infested (left) and infested (right) with psyllids in the greenhouse.

Diseased and healthy tubers were planted in greenhouse and field to determine if virus symptoms could be detected in the plants grown from such seed or in their progenies. A commercial seed stock of White Rose was secured which was known to contain a considerable number of affected tubers. Uncut tubers were held at room temperature until sprouting was well advanced, then separated into lots with normal and spindling sprouts. The tubers were cut into seed pieces with one sprout each and 20 hills of

each lot were planted in the field near Berkeley. All sprouts were held upright and were protected from damage while the soil was placed around them.

As soon as the spindling sprouts started to produce their own roots they began to lose their spindly characteristic. The apical portion of the sprouts thickened and soon after emergence the spindling sprouts closely resembled healthy sprouts. They were slower than normal sprouts, however, to appear above ground. This was in striking contrast to the growth of the sprouts in the laboratory where roots were absent and all nutrition was supplied by the seed piece. Without functional roots the spindling sprouts maintained a rather uniform diameter whether the growth was 2 inches or 2 feet in length. With functional roots, the spindling sprout, now an underground stem, eventually ceased to behave as a spindling sprout.

The foliage from these 2 lots of tubers was in every way comparable in appearance and in freedom from virus symptoms. The yield from the spindling sprout seed was about one-half that from the healthy seed. Tubers from these two lots were harvested and the progeny from each plant kept separate for a sprouting test. All progeny sprouted normally irrespective of whether they were derived originally from spindling sprout or normal tubers. These tubers were then replanted. The resulting plants were indistinguishable in appearance or in yield.

A smaller test of the same kind carried out in the greenhouse and two other field tests later gave similar results. For example, in one field test the yield from 10 blocks of normal seed potatoes containing an average of 18 hills per block was 266.5 pounds, whereas that from 10 blocks of spindling-sprout seed potatoes containing an average of 14 hills per block was 127.5 pounds. The average yield per hill was 1.5 pounds for normal seed potatoes and 0.8 pounds for spindling-sprout seed potatoes. In comparing these yields the reduction in stand obtained from the spindling-sprout seed and the resulting difference in spacings between hills must be taken into account.

All evidence gained from these tests supports the view that the spindling sprout studied is not caused by a virus. Spindling sprout was not transmitted to the progeny of plants grown from spindling-sprout seed, nor artificially by grafting. Furthermore, these tests showed that spindling sprout is a temporarily abnormal condition, recovery from which begins as soon as the diseased sprouts strike roots into the soil.

Toxin Indicated as a Cause. Since all available evidence was against a virus cause for the 1942 outbreak of spindling sprout in California and since no organism was found consistently associated with affected tubers, a toxic substance in the tuber itself was suspected. This hypothesis immediately suggested a possible relationship of spindling sprout to psyllid yellows, caused by *Paratrioza cockerelli* Sule.

In their paper on psyllid yellows of potato Richards and Blood (21) state that "tubers seldom reach marketable size and may sprout directly

without going into a rest period." They do not discuss the character of sprouting in tubers from psyllid infested plants, but state that the psyllid yellows disease is not tuber transmitted. Hartman (10) also observed in Wyoming that tubers from psyllid infested plants fail to go through a normal rest period and tend to sprout much earlier than normal tubers. Schaal (24) prepared an extract from crushed psyllids and injected it into potato plants. Not only did he report symptoms of psyllid yellows in the tops of these injected plants but noted that the tubers therefrom developed sprouts that were at first spindling. Schaal also made the incidental observation that psyllid infested plants produce tubers that give rise to normal plants except for spindly stems. That seed tubers from plants with severe vine injury caused by psyllids may develop weak stems and result in poor yields was shown also by Edmundson (6).

Experimentally Induced Spindling Sprout. In two greenhouse tests where a group of potato plants was intentionally infested with psyllids while another group was kept for controls, mild symptoms of spindling sprout were obtained when the tubers harvested from the infested plants had sprouted. Although well over a hundred psyllids were colonized on the infested plants, the work was done in the winter under poor light and even the foliage had very mild symptoms. However, since in these tests the noninfested controls produced only tubers that sprouted normally, the tendency for tubers from psyllid plants to produce spindly sprouts appeared significant. Moreover, a few tubers from this experiment developed typical spindling sprout.

In another experiment, 14 healthy potato plants of the White Rose variety were grown in 5-gallon cans in the greenhouse until tubers 1 to 2 inches long had set. Populations of over a hundred psyllids were at that time established on each of 7 plants. All plants were then moved outdoors into full summer sunlight, the controls being separated from the infested plants by a greenhouse. Typical, severe psyllid yellows symptoms appeared on the infested plants and the controls remained free from infestation.

The tubers were harvested separately from each plant and stored at room temperature until sprouted. Results are illustrated in figure 2. One or more tubers from each of the infested plants developed typical spindling sprout. Some tubers from infested plants never sprouted. Those which put forth sprouts did so well in advance of the tubers from the controls, and from more eyes per tuber. Every tuber from the control plants produced one to several normal sprouts. The resemblance of the spindling sprouts induced in this experiment to those obtained originally from the field or to those illustrated elsewhere (8, 11, 12, 14, 20, 28) is striking.

Before discarding the tubers from this experiment they were cut. Those harvested from psyllid-infested plants showed not only spindling sprout but also an internal browning resembling that reported by Sanford

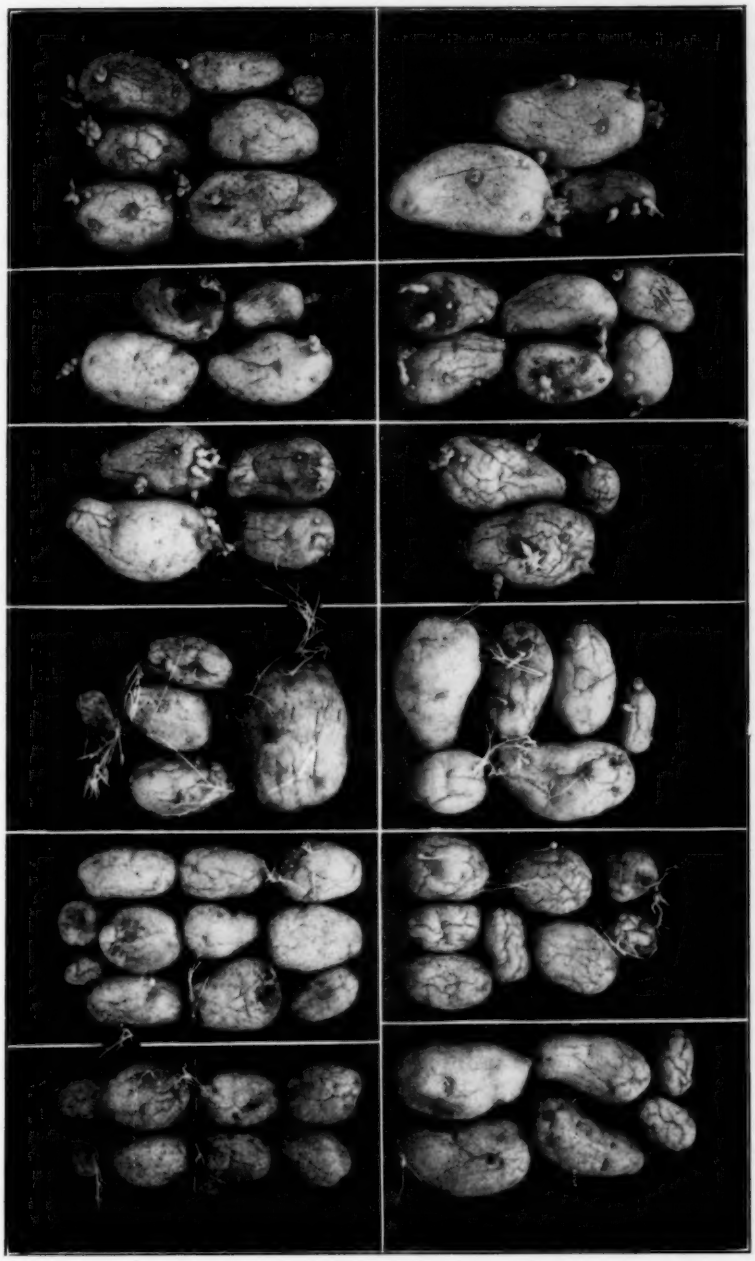

FIG. 2. Sprouted progeny from 6 noninfested plants (top) and 6 plants heavily infested (bottom) with psyllids, under outdoor conditions. Some affected tubers failed to sprout.

(23). These observations (27) confirm the suggestion that an infestation of the potato plant with psyllids may result in an internal necrosis of the tubers.

In another experiment an outdoor planting was made in midsummer and all plants were caged individually before plant emergence. To one group of plants, psyllids were introduced 2 months later. A second group was left noninfested for controls. A third group was infested with psyllids and at the same time the soil was inoculated with *Fasarium oxysporum* f. *tuberosi* Snyder and Hansen. A fourth group was inoculated with the *Fusarium* but not infested with psyllids. The result was that the progeny from all plants infested with psyllids developed typical spindling sprouts whether *Fusarium* was present or not, while all tubers from plants without psyllids sprouted normally.

DISCUSSION

Evidence presented here confirms the work of Schultz (26), Parris and Jones (20), and Harvey *et al.* (11) on one hand, and that of Richards and Blood (21), Hartman (10), and Schaal (24) on the other, that neither spindling sprout nor psyllid yellows is tuber transmitted. No involvement of a virus has been indicated in either case. That it is a toxic principle from the psyllid that produces both the foliage symptoms known as psyllid yellows and the symptom known as spindling sprout is supported by Schaal's (24) experiment in which he injected an aqueous filtrate of crushed psyllids into the potato plant.

Spindling sprout has been sporadic in its appearance, as have severe infestations of psyllids. Psyllids have been reported from Utah, Arizona, New Mexico, California, Washington, Oregon, Idaho, Montana, South Dakota, North Dakota, Kansas, Nebraska, and western Canada (10). Since psyllids are not conspicuous on a potato plant it is possible that a search may reveal their presence in still other potato-growing states. Relatively small numbers of psyllids on plants approaching maturity are probably sufficient in most cases to induce spindling sprout of the tubers.

It is possible that a toxicogenic insect other than psyllids may be responsible for non-virus spindling sprout in those regions where psyllids do not occur on the potato plant. If so, observations on the occurrence of spindling sprout in eastern states (1, 2, 8, 14, 28) would indicate an insect favored by a hot, dry growing season. In Iowa it has been observed (13) that more hair sprout has appeared in seasons immediately following years of severe purple top. This suggests that spindling sprout is a symptom of purple top as well as of psyllid yellows.

The prevention of spindling or hair sprout, where caused by the tomato psyllid, would be the same as that already worked out for psyllid yellows, namely, the frequent applications of sulphur dusts or sprays (4, 10, 22). In fact, recognition that psyllid infestations may result in the formation of spindling sprouts in the progeny provides an additional reason for psyllid control practices in seed potato regions.

SUMMARY

Artificial infestations of potato plants with psyllids under controlled conditions have shown that typical spindling sprout or hair sprout may be a symptom of psyllid yellows, when psyllids are present on plants approaching maturity.

The form of spindling sprout studied is not tuber transmitted, nor is there any evidence of the involvement of a virus.

There is no tendency for sprouts to grow out of the spindling sprout condition as long as all sprout growth is sustained by the tuber. Spindling sprouts that have struck roots into soil begin to grow normally.

The yield from spindling-sprout tubers was approximately one-half that from normal sprouting seed. The second generation from spindling-sprout tubers fully recovered from all spindling-sprout symptoms and yielded normally.

Severely affected tubers may fail to sprout.

Internal necrosis in tubers affected with spindling sprout was observed in one experiment in which the tubers were stored for several months.

The relation between spindling sprout and psyllid yellows makes doubly important the application of control practices against psyllids, especially in areas where seed potatoes are produced.

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LITERATURE CITED

1. ANONYMOUS. Potatoes. Maine Agr. Exp. Stat. Bull. 411–C. (1941–1942). pp. 302–3. 1942.
2. BARRUS, M. F., C. CHUPP, and K. H. FERNOW. Potato diseases and their control. N. Y. (Cornell) Agr. Exp. Stat. Bull. 135. 1926.
3. COONS, G. H. and J. E. KOTILA. Michigan potato diseases. Mich. Agr. Exp. Stat. Spec. Bull. 125. 1923.
4. DANIELS, LESLIE B. The tomato psyllid and the control of psyllid yellows of potatoes. Col. Agr. Exp. Stat. Bull. 410. 1934.
5. DYKSTRA, T. P. Potato diseases and their control. U. S. Dept. Agr. Farmers' Bull. 1881. 1941.
6. EDMUNDSON, W. C. The effect of psyllid injury on the vigor of seed potatoes. Amer. Pot. Jour. 17: 315–317. 1940.
7. EPPS, W. M. Purple top wilt of potatoes. Abstr. of Thesis, Cornell Univ. 1942: 363–365. 1943.
8. GIDDINGS, N. J. Potato and tomato diseases. West Va. Agr. Exp. Stat. Bull. 165. 1917.
9. GILBERT, ALFRED H. Correlation of foliage degeneration diseases of the Irish potato with variations of the tuber and sprout. Jour. Agr. Res. [U.S.] 25: 255–266. 1923.
10. HARTMAN, GLEN. A study of psyllid yellows in Wyoming. Wyoming Agr. Exp. Stat. Bull. 220. 1937.
11. HARVEY, R. B., A. REICHENBERG, BERNICE LEHNER, and P. C. HAMM. Hair sprout of potatoes. Plant Physiol. 19: 186–197. 1944.
12. JENSEN, J. H. and J. E. LIVINGSTON. Potato diseases in Nebraska. Neb. Agr. Exp. Stat. Bull. 378. 1945.
13. LONG, H. D. Purple top disease linked with hair-sprout tubers. Iowa State Hort. Soc. Rept. 70: 314–316. 1935.

14. MARTIN, WM. H. Potato diseases in New Jersey and their control. N. J. Agr. Exp. Stat. Circ. 146. 1923.
15. MCCOLLUM, J. P., M. B. LINN, and J. W. APPLE. Potato growing in Illinois. Ill. Agr. Exp. Stat. Circ. 583. 1944.
16. MCKAY, M. B., T. P. DYKSTRA, H. E. MORRIS, P. A. YOUNG, B. L. RICHARDS, and H. L. BLOOD. Virus and viruslike diseases of the potato in the northwest and their control. U. S. Dept. Agr. Circ. 271. 1933.
17. MICHENER, H. DAVID. An experiment on the physiological nature of spindling sprout. Proc. Amer. Soc. Hort. Sci. **42**: 511–513. 1943.
18. MORRIS, H. E. and M. M. AFANASIEV. Potato diseases in Montana and their control. Mont. Agr. Exp. Stat. Circ. 166. 1942.
19. PARISOT, F. La filosité de la pomme de terre. Bul. Mens. Off. Renseig. Agr. (Paris) **9**: 21–24. 1910.
20. PARRIS, G. K. and WINSTON W. JONES. Studies on the nature of spindling sprout of potato. Phytopath. **31**: 340–346. 1941.
21. RICHARDS, B. L. and H. L. BLOOD. Psyllid yellows of the potato. Jour. Agr. Res. [U.S.] **46**: 189–216. 1933.
22. RIEDL, WILLIAM A. Sulfur dusting for the control of psyllid yellows of potatoes. Wyo. Agr. Exp. Stat. Bull. 245. 1941.
23. SANFORD, G. B. and J. G. GRIMBLE. Observations on phloem necrosis of potato tubers. Can. Jour. Res., C, **22**: 162–170. 1944.
24. SCHAAL, LAWRENCE A. Some factors affecting the symptoms of the psyllid yellows disease of potatoes. Amer. Potato Jour. **15**: 193–206. 1938.
25. SCHULTZ, E. S. and DONALD FOLSOM. Leafroll, net-necrosis, and spindling-sprout of the Irish potato. Jour. Agr. Res. [U.S.] **21**: 47–80. 1921.
26. SCHULTZ, E. S. A non-transmissible spindling sprout of potato. (Abstr.) Phytopath. **29**: 21. 1939.
27. SNYDER, WILLIAM C., H. EARL THOMAS, and S. J. FAIRCHILD. A type of internal necrosis of the potato tuber. Phytopath. **36**: 480–481. 1946.
28. STEWART, F. C. and F. A. SIRRINE. The spindling-sprout disease of potatoes. N. Y. (Geneva) Agr. Exp. Stat. Bull. 399. 1915.
29. YOUNKIN, S. G. Purple-top wilt of potatoes caused by the aster yellows virus. Amer. Pot. Jour. **20**: 177–183. 1943.

BLACK ROT OF MUSCADINE GRAPES[1]

E. S. LUTTRELL

(Accepted for publication July 10, 1946)

Black rot, a widespread and destructive disease of bunch grapes, is also a common disease of muscadine grapes, *Vitis rotundifolia* Michx. On muscadine grapes it is primarily a disease of the vegetative parts; while on bunch grapes it is primarily a disease of the fruit, mummifying the berries and often destroying a large part of the crop. In addition to differences in symptoms and in amount of damage to the crop the black rot disease of muscadine grapes differs from that of bunch grapes in etiology. The disease as it occurs on muscadine grapes has, however, received only incidental consideration, although a great amount of work has been done on black rot of bunch grapes. Since the muscadine grapes are now becoming of increased economic importance, it seems desirable to present an account of work done during the past four years at the Georgia Experiment Station on the symptoms, etiology, and control of the black rot disease on these grapes and to attempt an estimate of the effect this disease may have upon the production of the crop in the future.

SYMPTOMS

Black rot is common in muscadine vineyards in Georgia throughout the growing season. In fact, from April to late August, it is the only disease of any importance found in these vineyards. Other diseases become prevalent only toward the end of August as the berries approach maturity. Angular leaf spot (3) then appears on the foliage, and bitter rot and various ripe rots develop on the maturing berries. All of these diseases may be easily distinguished from black rot. Nevertheless, because of the superficial resemblance of bitter-rot berry mummies to the berry mummies produced by black rot on bunch grapes, bitter rot and black rot of muscadine grapes have sometimes been confused by growers. The two diseases should be distinguished, however, as black rot usually causes only a scab or canker of muscadine berries, while bitter rot reduces the berries to shriveled black mummies.

On Leaves. The first symptoms of black rot are leaf spots which appear almost invariably each year about four weeks after the shoots begin their annual growth. The spots appear suddenly throughout the vineyard and attain their full size almost as soon as they become evident. They are

[1] Paper No. 149, Journal Series, Georgia Experiment Station, Experiment, Georgia.

Work on diseases of muscadine grapes was turned over to me in October, 1942, by Dr. K. H. Garren. Dr. F. F. Cowart, Horticulture Department, Georgia Experiment Station, has collaborated on this project to the extent of applying sprays in experiments on control and furnishing data on yields.

I am indebted to Dr. B. B. Higgins, Dr. F. F. Cowart, Dr. K. H. Garren, Georgia Experiment Station, Dr. J. L. Weimer, Bureau of Plant Industry, and Dr. Frederick A. Wolf, Duke University, for their criticisms of the manuscript and to Mr. J. G. Futral for taking the photograph reproduced in figure 3, B.

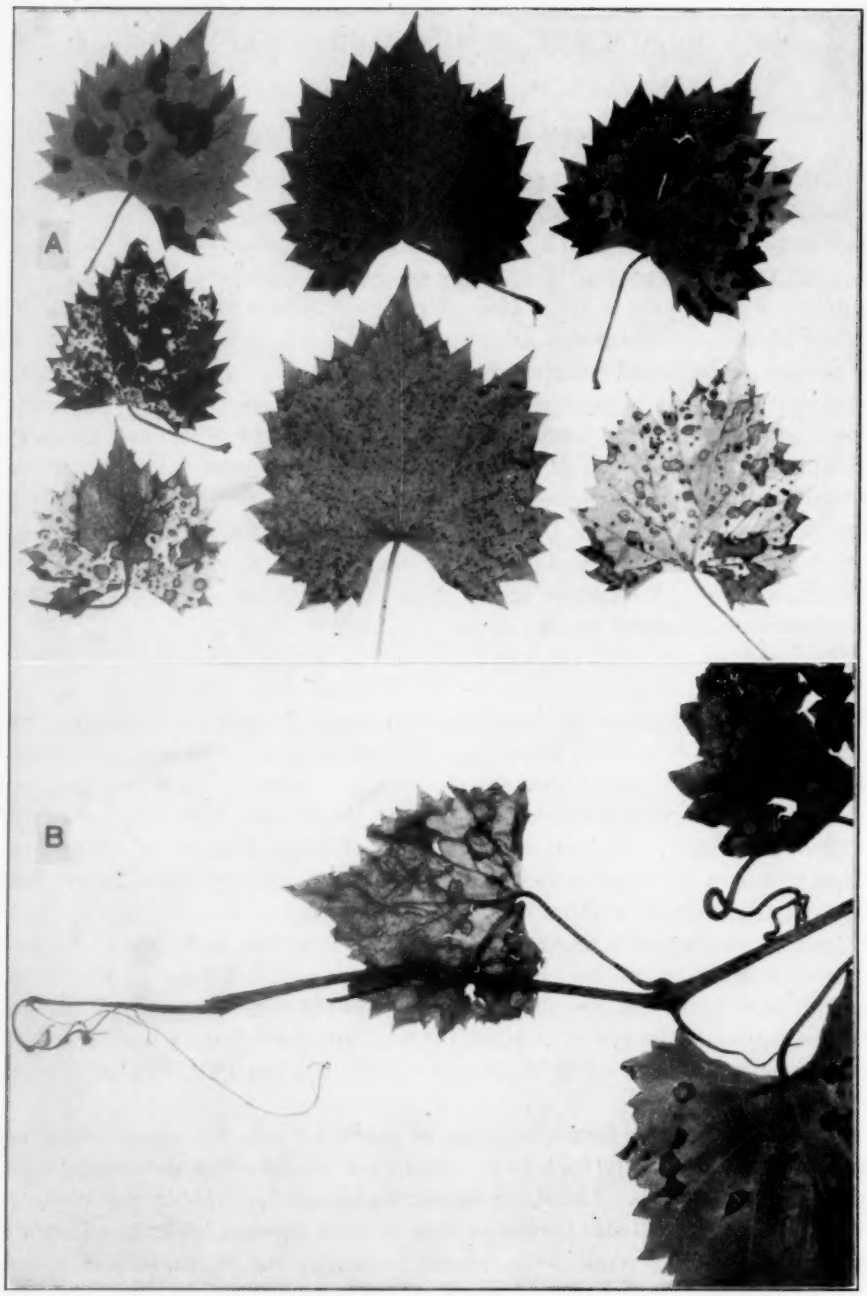

FIG. 1. A. Black-rot spots on leaves of a muscadine grape; upper four leaves from the upper surface, lower three leaves from the lower surface; $\times \frac{3}{5}$. B. Black-rot stem tip blight of a muscadine grape shoot; $\times \frac{1}{4}$.

first noticeable as faint blanched areas in the leaf blade. The green color of the infected tissue fades; and the spots become cream-colored, the color gradually deepening to tan and ultimately on the upper surface to reddish-brown. They are usually bordered by a narrow band of dark brown tissue. Pycnidia develop within two or three days on both surfaces of the leaf. When mature they appear as small black pimples in the dead brown tissue of the spots. The spots are circular and vary in diameter from less than one to 12 mm. (Fig. 1). Large spots may coalesce and kill a large part or all of the leaf blade (Fig. 1). Infection of major veins also may result in the death of large areas of the leaf (Fig. 1). Mature leaves are not infected.

Petioles may become infected at the same time as the leaf blades. Either one to several small, scabby, flat or slightly elevated black lesions or larger, depressed cankers develop in the petioles. Small lesions apparently do not interfere with the functioning of the petiole, but large cankers may girdle the petiole and kill the entire leaf (Fig. 2, A).

Infection of immature leaves continues to occur during periods of favorable weather throughout the season until growth of the vines ceases in late fall. Generally, however, black rot causes little defoliation.

On Stems. Infection of the stems just back of the growing tip occurs throughout the growing season. Either small, black, scabby lesions (Fig. 2, A) or elongated, black, depressed cankers (Fig. 2, B) are produced in the surface of the stem. Numerous cankers sometimes cause a blight of the growing tips of the shoots. The stem becomes blackened and dies, the leaves drop off, and the tip of the stem curves back to form a crook (Fig. 1, B). Stem tip blight is especially common after periods of moist weather in spring. Like the stems, tendrils may be more or less covered with cankers or even killed.

On Flower Clusters. Black rot also affects the flower clusters. Cankers similar to those on the petioles appear on the peduncles and pedicels (Fig. 2, A). Small cankers apparently cause little damage, but more extensive cankers may kill a portion or all of the cluster (Fig. 2, A). Blight occurs chiefly on the first flower clusters produced in the spring. Those formed later in the season are rarely blighted. Moreover, the cankers apparently do not spread in the pedicels and peduncles which survive infection.

On Berries. On most varieties of muscadine grape only one to several small, black, superficial, scabby lesions one to two mm. in diameter are formed in the skin of the berry (Fig. 3, A, row 3, No. 2–5). Occasionally, especially on the Dawn variety, the scabs may coalesce to form a brown to black mosaic-like crust covering a large part of the surface of the berry (Fig. 3, A, row 3, No. 1). Cankers up to 10 mm. in diameter may be produced on the berries of the most susceptible varieties (Fig. 3, A, row 2, No. 6–7). The skin often splits at the periphery of these cankers, exposing a band of brownish corky tissue which forms a border around the canker. The surface of the canker is cracked and fissured and is roughened with embedded pycnidia. Infection takes place on the immature berries from

the time they are set until they approach full size. The lesions do not spread in mature berries, and these dry, superficial scabs and cankers usually do not cause the berries to drop.

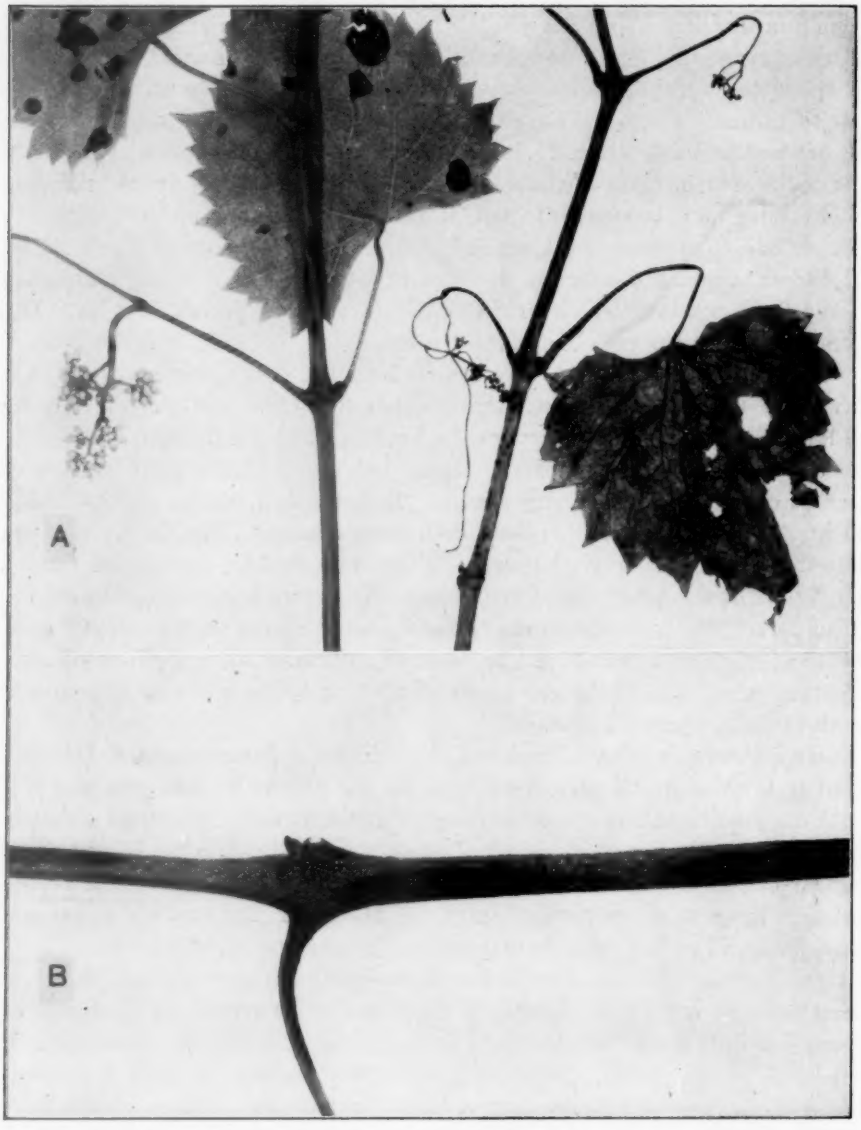

FIG. 2. A. Black-rot blossom blight of a muscadine grape. Several peduncle cankers are on the flower cluster at the left. The two flower clusters on the stem at the right are entirely blighted. The leaf at the right is also blighted as the result of severe cankers on the petiole; × ¾. B. Black-rot cankers on the stem of a muscadine grape; × 1½.

Black rot does, however, cause a small amount of berry drop on the most susceptible varieties such as Hunt and November in seasons that are

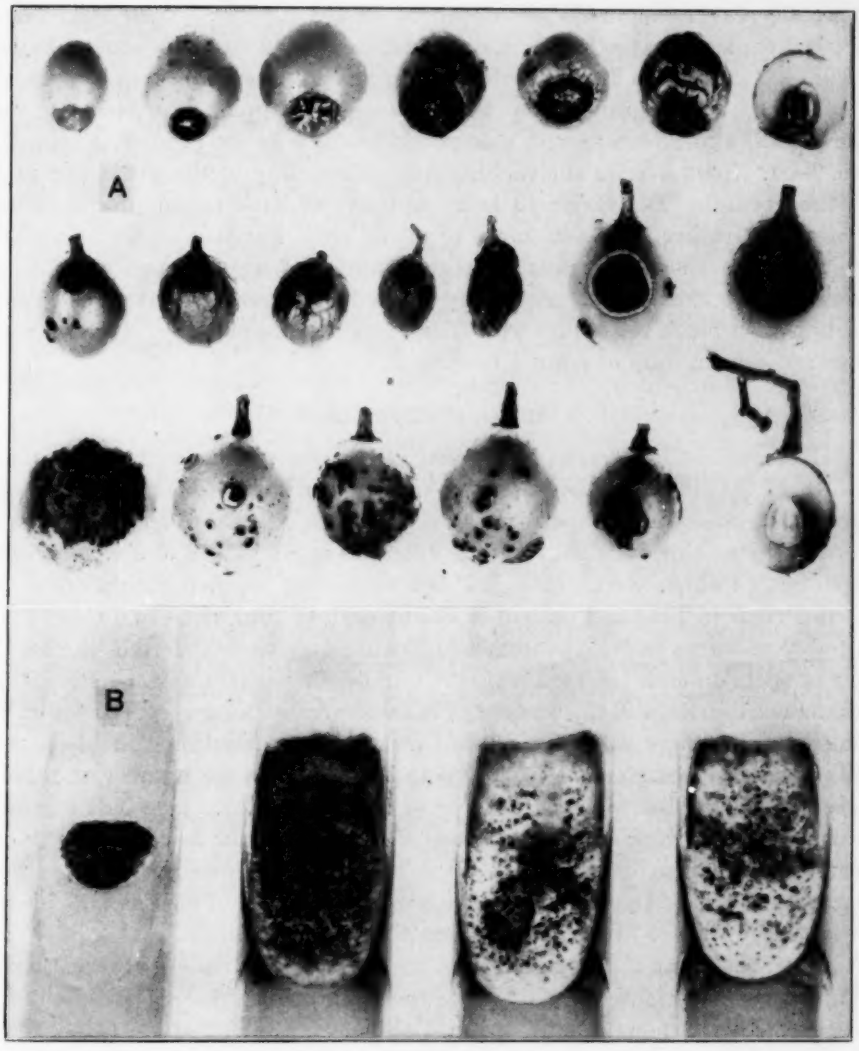

FIG. 3. A. Black-rot scabs and cankers on muscadine berries. Row 1, left to right, a series of berries of the Hunt variety showing the development of a severe canker: the three berries on the right had dropped from the vine; the berry on the extreme right is sectioned to show the deep canker extending inward to the seed. Row 2, berries 1–5, berries of the Hunt variety showing the formation of a shriveled "raisin" resulting from the development of a canker at the point of attachment to the pedicel; these shriveled berries were clinging to the vine; berries 6 and 7, large cankers on berries of the same variety. Row 3, berry 1, a berry of the Dawn variety showing a mosaic-like crust resulting from infection with black rot; berries 2–6, superficial scabs on the surface of Hunt berries; the berry at the extreme right has been sectioned. ×1.

B. *Guignardia bidwellii* in culture tubes on 3 per cent malt agar; all colonies are the same age; left to right, *G. bidwellii* var. *muscadinii* isolated from ascospores from a muscadine grape leaf, *G. bidwellii* isolated from ascospores from berry mummies of a Warren bunch grape, of *Vitis vinifera*, and of an unknown variety of eastern bunch grape. ×1.

especially favorable to the development of the disease. Then large, depressed cankers that extend inward to the seed may form on some berries (Fig. 3, A, row 1). These berries shrivel slightly and fall from the vines. Cankers also may form on the berry close to the point of attachment to the pedicel and interfere with conduction into the berry (Fig. 3, A, row 2, No. 1–5). Such berries shrivel and may either cling to the pedicel or fall to the ground. The shriveled berry appears to be a raisin produced by desiccation rather than a mummy resulting from invasion of the berry by the fungus. The canker does not spread, and the berry becomes reddish or brown rather than black. Any drop of the berries resulting from their infection with black rot occurs when the berries are immature. This disease does not cause a drop of mature berries.

VARIETAL SUSCEPTIBILITY

Observations in a variety vineyard at the Georgia Experiment Station indicated that there was considerable variation in susceptibility to black rot among the muscadine grape varieties represented. These observations were tested by counting infected leaves and berries on 16 named and 8 unnamed varieties. Counts were made just before harvest on five shoots on each of two vines in 1943 and on five shoots on each of four vines in 1944. The number of leaves fallen, the number of remaining leaves infected, the number of spots on each infected leaf, the number of cankers on each stem, the number of berries, and the number of berries infected were recorded for each shoot. Since there was only a small amount of defoliation and black rot was the only disease on the leaves up to harvest time, the number of fallen leaves was included with the number of leaves infected. The best criteria of susceptibility seem to be percentage of berry infection and percentage of leaf infection plus defoliation. Upon the basis of these two criteria the varieties may be separated into three general groups and classified as being very susceptible, of intermediate susceptibility, or resistant.

Upon the basis of berry infection, the varieties November, Howard, and Hunt were very susceptible (24–18 per cent infection); Dawn, Creek, Thomas, Scuppernong, Spalding, and LaSalle were intermediate (11–3.5 per cent infection); and Stuckey, Brownie, Lucida, Big Boy, Irene, Dulcet, and Yuga were resistant (2–0.35 per cent infection). These results on susceptibility of berries were checked in 1945, a season in which diseases were exceptionally severe, by making counts on several of the most susceptible and the most resistant varieties. Fifty berry clusters on each of two vines of each variety were counted, and the percentage of berries infected was recorded. The results were as follows: Hunt, 861 berries—29.27 per cent infected; Howard, 494 berries—16.60 per cent infected; Dawn, 500 berries—14.40 per cent infected; Thomas, 776 berries—1.16 per cent infected; and Dulcet, 825 berries—0.24 per cent infected. The berries of resistant varieties, when they are infected; usually bear only small, superficial scabs; while the berries of susceptible varieties are often severely cankered. This difference in severity of cankers was demonstrated in 1945 by collecting

at weekly intervals from the first of August until after harvest all of the berries that fell to the ground from two vines of each variety and recording the percentage of fallen berries that dropped because of black-rot cankers. Berry drop was severe on all varieties, but little of it was caused by black-rot berry cankers. Cankers caused berry drop on only six varieties, three of which were those classed as very susceptible. The percentages of fallen berries that dropped because of black-rot cankers are as follows: November, 2.34; Hunt, 2.26; Howard, 0.72; Creek, 0.24; Dawn, 0.18; and Spalding, 0.17.

Upon the basis of leaf infection plus defoliation Stuckey, Dawn, Howard, Scuppernong, LaSalle, November, and Hunt were very susceptible (70–60 per cent infection); Brownie, Spalding, Big Boy, Lucida, Yuga, Creek, and Irene were intermediate (59–50 per cent infection); and Dulcet and Thomas were resistant (40–37 per cent infection). While the majority of the varieties are resistant to infection of the berries, most of them are susceptible to infection of the leaves. Furthermore, resistance of the berries is not necessarily correlated with resistance of the foliage. For example, the Stuckey variety, whose berries are resistant, has extremely susceptible foliage.

Upon the basis of both leaf infection and berry infection Dulcet seems to be the most resistant of these 16 varieties. Thomas, Yuga, Irene, and Big Boy also show a considerable degree of resistance. November, Howard, and Hunt appear to be the most susceptible varieties.

IMPORTANCE

Exact data on the effect of black rot on vegetative parts of muscadine grapes could be obtained only by comparing yields of infected vines with yields of vines on which the disease was completely controlled. Control of the disease on vegetative parts has, however, proved too difficult to make possible such a comparison. In the absence of these data only an estimate of the importance of the black-rot disease can be made. Although the percentage of leaves remaining at harvest time on the vines which are infected is high in all varieties, varying from 33 per cent on Thomas to 65 per cent on Stuckey, the amount of defoliation, not all of which can be attributed to black rot, is low. On most varieties it is less than 10 per cent. On Thomas and Dulcet it is approximately 6 per cent, and on Yuga and Creek it is approximately 4 per cent. Exceptions are Stuckey and Scuppernong with 18 and 17 per cent defoliation, respectively. Although a great many leaves are diseased, such leaves generally continue functional. On all varieties the proportion of stems infected averages 63 per cent. Only 2 per cent of the stems are blighted at the tips, however; and axillary buds continue the growth of blighted shoots almost immediately. In view of the fact that muscadine grapes are extremely vigorous, producing an excess of vegetative growth, it seems improbable that either the reduction in functional leaf area as a result of spotting or the small amount of stem tip

blight is of much importance in a properly managed vineyard. Only Stuckey and possibly Scuppernong, on which there is a great amount of leaf infection and a considerable amount of defoliation, appear to be affected by the presence of black rot on the vegetative parts.

Blossom blight appears to be of more importance than leaf infection although here again, since control has not been obtained, no exact data on its influence on yield are available. Blossom blight is occasionally severe on the first flower clusters formed in the spring. There is little blossom blight later in the season. Since muscadine grapes bloom over a long period, it is possible that fruit set late will compensate for that lost as a result of blight of the flower clusters early in the spring. But, even so, severe blossom blight results in irregular ripening of the crop, and this is objectionable because it increases the cost of harvesting.

On most varieties berry infection is negligible. Berry infection must be considered, however, because Hunt, the variety of greatest promise for commercial growers, is one of the most susceptible varieties. Infection of Hunt berries may be as high as 30 per cent, and the cankers may be severe enough to cause the berries to drop. Even on Hunt, however, berry drop resulting from infection with black rot is of little importance. In 1945 thirty-five per cent of the crop on Hunt vines in the Experiment Station variety vineyard, dropped to the ground. Bitter rot, the most important single factor, accounted for 55 per cent of this drop. Black-rot cankers on the berries were responsible for only 2 per cent of it. This represents only 0.4 lb. lost because of black rot of a total potential yield of 79 lb. per vine. Since black-rot cankers do not spread in mature tissue, it is improbable that cankers on the pedicels were responsible for any considerable amount of the berry drop. Black rot is, therefore, considered of little importance in reducing yields. This is illustrated by the results from spraying. In 1943 counts of infected berries on Hunt vines showed that 30.7 per cent of the berries were infected on unsprayed vines, while only 14.69 per cent were infected on sprayed vines. This reduction of approximately 50 per cent in amount of infection was not, however, reflected in increased yields. On both the sprayed and the unsprayed vines the average yield was 48 lb. per vine. The belief that black rot is not of primary importance in influencing yield is further substantiated by the fact that, during the past six years in the Experiment Station variety vineyard, the Hunt variety has produced an average annual yield of 66.17 lb. per vine; while Dulcet, the most resistant variety, has produced only 31.17 lb. per vine.

Black rot does, however, affect the quality of the crop. Although the superficial, scabby lesions do not reduce the yield, they damage the appearance of the fruit.

ETIOLOGY

Taxonomy. Observation, first by Dr. B. B. Higgins, of the fact that bunch grape vines growing adjacent to or intertwined with heavily infected

muscadine vines often remain free of black rot suggested that the fungus associated with black rot of muscadine grapes differs in pathogenicity from *Guignardia bidwellii*, the cause of the disease on bunch grapes. Results of inoculation trials have now confirmed this observation. Because this difference in pathogenicity is accompanied by only slight morphological differences, the fungus causing black rot of muscadine grapes is described as a new form of *G. bidwellii*.

Guignardia bidwellii f. muscadinii f. nov.

Status perithecialis: Peritheciis nigris, amphigenis, plerumque hypophyllis, punctiformibus, per totum folium dispositis, subinnatis, sphaeroideis, 61.6–112.0 μ (medio. 84.28 μ diam., poro praeditis; ascis fasciculatis, aparaphysatis, cylindraceis vel clavatis, 36.4–56.0 × 11.76–16.8 μ (medio 43.76 × 14.28 μ), sessilis vel breve stipitatis, bitunicatis, octosporis; ascosporis hyalinis, continuis, obovatis vel oblongis, rectis vel plerumque leniter inaequalibus, 13.5–17.25 × 6.0–9.0 μ (medio, 14.92 × 7.24 μ), biseriatis vel inordinatis.

Hab. in verno in foliis dejectis *Vitis rotundifoliae* Michx., Experiment, Georgia.

Status spermogonicus: Spermogoniis nigris, punctiformibus, amphigenis, plerumque hypophyllis, cum stromatibus carpogonialibus per totum folium dispersis, subinnatis, globosis, 44.8–78.4 μ diam., poro praeditis; spermatiis hyalinis, bacillaribus, plerumque 2.5 × 1 μ, intus loculo centrali efformantis.

Hab. in autumno in foliis dejectis *Vitis rotundifoliae*.

Status conidicus: Pycnidiis nigris, punctiformibus, amphigenis in foliis in maculis rufo-brunneis, orbicularibus, 1–12 mm. lat. vel in ramis et fructibus in laesionibus nigris effortmantis, globosis, 58.8–126.0 μ diam., poro praeditis; pycnosporis hyalinis, non-septatis, obovatis, oblongis, vel subglobosis, utrimque obtusculis, 7.98–13.30 × 5.32–7.5 μ (medio 10.16 × 6.40 μ), ex hyphis brevis intus loculo centrali orinndis.

Hab. in verno, aestivo, atque autumno in laesionibus in foliis, ramis, et fructibus vivis *Vitis rotundifoliae*.

Collections of type material have been deposited in the Farlow Herbarium, Harvard University, in the Mycological collections of the Bureau of Plant Industry, U. S. Department of Agriculture, and in the herbarium of the Royal Botanic Garden at Kew, Surrey, England.

The form *muscadinii* differs from *Guignardia bidwellii* in pathogenicity, in appearance and rate of growth of colonies in culture, in the smaller size of perithecia, and in the slightly larger size of ascospores and pycnospores. These differences will be brought out in detail in the following account. They are not considered sufficient basis for the erection of a new species or variety.

Morphology. Only the pycnidial stage of *Guignardia bidwellii* f. *muscadinii*, which belongs in the form genus *Phoma*, is associated with black-

rot lesions on muscadine vines during the growing season. Primary infection of the leaves and stems as soon as they emerge from the buds in the spring is accomplished by ascospores from perithecia on overwintered leaves and by pycnospores from cankers on stems infected during the previous season. The spores are carried by air currents or raindrops to the surface of the new growth where, under suitable moisture conditions, they germinate. Although entrance of the germ tubes has not been observed directly, evidence obtained from inoculations indicates that infection takes place through the upper surface of the leaf as readily as through the lower surface. Since the upper epidermis of the leaf is not provided with stomata, the germ tubes must be capable of penetrating the epidermis directly. Within the leaf the mycelium spreads intracellularly through the epidermis and mesophyll without producing externally visible symptoms for about three weeks. At the end of the incubation period the invaded cells die, producing the characteristic black-rot lesions. A similar invasion of immature tissue of petioles, stems, tendrils, flower clusters, and berries results in the production of cankers. The cells of the mycelium filling the dead epidermal cells become thick-walled and dark-colored, producing a black crust over the surface of the canker. On berries the infected tissue is separated from the healthy tissue beneath by layers of cork cells.

Pycnidia develop rapidly within the lesions as the infected tissue dies. The pycnidium originates as a spherical mass of pseudoparenchymatous tissue beneath the epidermis. Within the pycnidium a central cavity is formed, and cells lining the cavity produce short conidiophores which abstrict pycnospores from their tips. The apex is provided with a pore through which the pycnospores which now fill the cavity are liberated. The pycnospores are sub-globose, obovate, or oblong, hyaline, and nonseptate. They immediately initiate secondary infections.

The fungus survives the winter on infected stems and leaves. No spores of *Guignardia bidwellii* f. *muscadinii* have been found on overwintered berries. This may be because the berries, as soon as they fall to the ground, are invaded by various fungi that are more vigorous saprophytes. Functional pycnidia survive the winter in cankers on the stems, but these cankers are excised by growth of the stems during the second season. Pycnospores have also been found in abundance on infected stems which were pruned off in the fall and left lying on the ground over winter.

Pycnidia on leaves do not survive the winter, but perithecia are produced abundantly on over-wintered leaves. After the leaves die and fall from the vines at the end of the growing season, the fungus spreads throughout the leaf. From October to December, spermogonia and perithecial initials develop in the dead leaves. They are formed over the entire surface of the leaf outside of the original lesions. Externally spermogonia are similar to pycnidia except for their smaller size. The spermogonium is composed of a pseudoparenchymatous wall surrounding a central cavity filled with hyaline, unicellular, bacilliform spermatia. The spermatia are liberated through a pore in the apex of the spermogonium.

Perithecial initials develop slightly later than spermogonia but are externally indistinguishable from them. They differ internally, however, in that no central cavity is formed. The perithecial initial is filled with a mass of large, thin-walled, hyaline pseudoparenchymatous cells. Several deeply-staining carpogonial cells lie in the pseudoparenchyma within each perithecial initial. From these cells a fascicle of asci is produced in the base of the developing perithecium during January. As the asci develop the pseudoparenchyma is crushed and disintegrated leaving a central cavity occupied by the asci. The ascospores mature during late March. The mature perithecium consists of a spherical, dark-colored, pseudoparenchymatous wall surrounding a central cavity containing a fascicle of aparaphysate asci. At the apex the wall is provided with a pore through which the ascospores are discharged. Mature asci are sessile or short-stipitate and are cylindrical to clavate. Each contains eight biseriately arranged ascospores. The ascospores are obovate to oblong, hyaline, and non-septate. When the asci are moistened, they discharge their ascospores forcibly one after another. Ascospore discharge continues for four or five weeks during April and May.

Guignardia bidwellii and its form *muscadinii* differ in symptoms produced on the host plants as well as in morphology. On bunch grapes infection with *G. bidwellii* results in the complete mummification of the berries, while on muscadine grapes *G. bidwellii* f. *muscadinii* causes only a superficial scab or canker of the berries. It is quite possible, however, that this difference in symptoms is the result of host reaction. The differences in morphology also are slight (Table 1). Perithecia of *G. bidwellii* were larger than those of the form *muscadinii*, the difference in average diameter being 62.2 μ. Since perithecia of *G. bidwellii* are produced only on berries and those of the form *muscadinii* only on leaves, the larger size of the perithecia of *G. bidwellii* may be a result of growth on a richer substrate. This will not, however, explain the difference in size of ascospores since the ascospores of the variety were 1.4 μ longer and 0.6 μ wider than those of the species. There was little difference in size of pycnidia. The pycnospores of the form were slightly longer than those of *G. bidwellii*, the difference being 1.0 μ in average length. A similar difference was apparent in the size of pycnospores produced on leaves of *Vitis vinifera* inoculated with the two fungi. On the same host the difference in average length of pycnospores was 0.8 μ.

Growth in Culture. *Guignardia bidwellii* f. *muscadinii* was obtained in culture from ascospores by suspending pieces of over-wintered muscadine leaves bearing mature perithecia from the top of a Petri dish so that the ascospores were discharged upon the agar below. Cultures were obtained also from infected tissue of muscadine stems, leaves, and berries by planting bits of surface-sterilized tissue cut from the margins of black-rot lesions on tapwater agar. Isolates from all sources produced similar colonies on 3 per cent malt agar. The colonies (Fig. 3, B) were slow-growing,

TABLE 1.—*A comparison of the morphology of Guignardia bidwellii and its form muscadinii*

Part	*G. bidwellii*		*G. bidwellii* form *muscadinii*	
	On bunch grape		On muscadine grape	
Perithecia	95.2–198.8 μ	(av. 50 = 146.5 μ)	61.2–112.0 μ	(av. 50 = 84.3 μ)
Ascospores	11.3–16.5 × 6.0–6.8 μ	(av. 100 = 13.5 × 6.6 μ)	13.5–17.3 × 6.0–9.0 μ	(av. 100 = 14.9 × 7.2 μ)
Pycnidia				
(on leaf)	61.6–95.2 μ	(av. 25 = 79.4 μ)	58.8–106.4 μ	(av. 25 = 82.9 μ)
(on berry)	114.8–196.0 μ	(av. 25 = 145.4 μ)	70.0–126.0 μ	(av. 25 = 97.9 μ)
(on stem)	98.0–137.2 μ	(av. 25 = 113.2 μ)	61.6–117.6 μ	(av. 25 = 91.4 μ)
Pycnospores	7.5–11.5 × 5.6–8.0 μ	(av. 100 = 9.2 × 6.7 μ)	8.0–13.3 × 5.3–7.5 μ	(av. 100 = 10.2 × 6.2 μ)
	On vinifera grape		On vinifera grape	
Pycnidia	68.0–149.6 μ	(av. 25 = 113.2 μ)	68.0–149.6 μ	(av. 25 = 97.5 μ)
Pycnospores	8.0–12.0 × 6.4–6.7 μ	(av. 25 = 8.5 × 6.6 μ)	8.0–13.3 × 6.0–6.7 μ	(av. 25 = 9.3 × 6.3 μ)

black, hemispherical masses of plectenchymatous stromatic tissue densely covered with pseudoparenchymatous pycnidia. Pycnospores, produced abundantly in these cultures after three to four weeks, were identical with those found in black-rot lesions on muscadine vines. Pycnospore production ceased after several weeks, and no pycnospores were formed in transfers of the original cultures. In transfers a scanty white mycelium developed over the colonies, partially covering the black stromatic mass and extending slightly beyond its margin.

For comparison, *Guignardia bidwellii* was isolated from ascospores from perithecia on over-wintered black-rot berry mummies from the following three different bunch grapes: (1) a European grape near Conyers, Georgia, (2) an undetermined variety of native grape at the same locality, and (3) a Warren grape near Experiment, Georgia. The isolates from all three sources were similar to one another but different from those of the form *muscadinii*. On 3 per cent malt agar the three isolates produced more rapidly growing colonies of white mycelium which soon became gray or black at the surface of the medium (Fig. 3, B). Pycnidia were scattered separately in the mycelium. The pycnospores formed in these cultures were similar to those formed by *G. bidwellii* f. *muscadinii*. Pycnospores were again produced in the first transfers from the original cultures, but they were not formed in subsequent transfers.

In addition to the differences in type of colony formed by *Guignardia bidwellii* and its form *muscadinii* and in rates of growth in culture there was a slight difference in size of pycnospores produced by the two fungi under identical conditions on the same culture medium. The pycnospores of *G. bidwellii* were $7.5–12.0 \times 6.0–9.0$ μ (av. 125, 9.17×6.97 μ); those of the form *muscadinii* were $7.80–14.63 \times 5.32–9.31$ μ (av. 125, 10.09×6.92 μ). Although there was no difference in width, the pycnospores of *G. bidwellii* f. *muscadinii* exceeded those of *G. bidwellii* both in maximum length and in average length. The difference in maximum length was 2.63 μ, while the difference in average length was 0.92 μ.

Inoculations. Inoculation experiments were designed to determine (1) the pathogenicity of *Guignardia bidwellii* and *G. bidwellii* f. *muscadinii* on several species and varieties of *Vitis,* (2) the differences in pathogenicity between the species and its form, and (3) the genetic connection of the perfect and the imperfect stages of both fungi. All inoculations were made in the greenhouse where there was no natural infection. The inoculum used was suspensions of pycnospores obtained by macerating pycnidia from cultures or from host lesions in sterile tap water. Host material was furnished by small vines grown in 6- or 8-inch pots. In making inoculations single, vigorously growing shoots bearing leaves in various stages of development were selected. The number of leaves on each shoot and the size of each leaf were recorded. Drops of inoculum were applied along the stem and to each leaf on the shoot. After permitting the drops of inoculum to dry, the inoculated plants were placed in a moist chamber for 48 hours.

The plants were then returned to benches in the greenhouse and kept under observation for at least one month. Control plants included in each experiment were treated similarly except for the fact that they were not inoculated.

Only the muscadine grape was inoculated with pycnospores from field material of *Guignardia bidwellii* and *G. bidwellii* f. *muscadinii* in July, 1943. Six shoots bearing a total of 70 leaves were inoculated with pycnospores of *G. bidwellii* f. *muscadinii* obtained from leaf spots on muscadine vines. All six shoots became infected. Leaf spots or petiole cankers or both appeared on 31 of the 70 leaves. Five of the leaves were killed. The stems of two of the shoots were cankered, and their tips were blighted. Eleven control shoots with 85 leaves remained free of infection. Eight shoots with 64 leaves were inoculated with pycnospores of *G. bidwellii* from leaf spots on a Warren bunch grape, and six shoots with 49 leaves were inoculated with pycnospores from leaf spots on an undetermined species of wild bunch grape. None became infected. In these experiments *G. bidwellii* f. *muscadinii* produced 100 per cent infection on muscadine shoots; while *G. bidwellii* produced no infection on similar shoots.

During June in 1944 and 1945 more extensive inoculations with both *Guignardia bidwellii* and *G. bidwellii* f. *muscadinii* were made on four different hosts, (1) a European bunch grape (*Vitis vinifera* Linn.), (2) a muscadine grape (*V. rotundifolia*), (3) a Warren bunch grape (*V. bourquina* Munson ex Viala), and (4) a Niagara bunch grape (*V. labrusca* Linn.). Inoculum of *G. bidwellii* f. *muscadinii* was a suspension of pycnospores produced in cultures of ascospores from over-wintered muscadine leaves. Inoculum of *G. bidwellii* was suspensions of pycnospores produced in cultures of ascospores from over-wintered berries of three different bunch grapes; a European, a Warren, and an undetermined variety of native grape. Since inoculum of *G. bidwellii* from these three different sources produced similar results, they will not be considered separately. The results of these inoculations are in table 2. During these experiments, the control shoots remained free of infection. *G. bidwellii* f. *muscadinii* produced 100 per cent infection of muscadine shoots but did not infect Warren or Niagara shoots. On the other hand, while *G. bidwellii* infected almost 90 per cent of the Warren and Niagara shoots, it did not infect muscadine shoots. Both, however, produced 100 per cent infection of vinifera shoots. The two fungi were reisolated from lesions on the inoculated shoots by planting bits of surface-sterilized infected tissue on tap-water agar. Both *G. bidwellii* and the form *muscadinii* were reisolated unchanged from the vinifera grape.

The incubation period of *Guignardia bidwellii* on all bunch grapes was approximately two weeks. The incubation period of *G. bidwellii* f. *muscadinii* on muscadine grapes was approximately three weeks. On the vinifera grape, however, its incubation period was two weeks, the same as that of *G. bidwellii*. The longer incubation period of the form *muscadinii* on muscadine grapes appears, therefore, to be the result of host reaction, and it does not constitute a physiological difference between *G. bidwellii* and its variety.

The low percentage of leaf infection (Table 2) may be explained by the fact that only immature host tissue is susceptible. If only immature leaves were selected for inoculation, the percentage of leaves infected might approach 100 per cent. For this reason the differences in amount of leaf infection are not considered significant. They are simply a result of variation in the shoots inoculated, young rapidly growing shoots presenting a greater proportion of immature susceptible leaves than older shoots.

On muscadine vines spots appeared only on the first to the seventh leaf back of the growing tip. These leaves at the time of inoculation varied from tiny leaves not yet expanded to leaves up to 4.7 cm. across. No spots ever appeared on larger, more nearly mature leaves farther back from the tip of the shoot. Apparently the fungus spread within the leaf tissue without

TABLE 2.—*Results of inoculation trials with Guignardia bidwellii on grapes*

Hosts	*G. bidwellii*					*G. bidwellii* f. *muscadinii*				
	Shoots inoculated	Shoots infected	Leaves inoculated	Leaves infected	Stems infected	Shoots inoculated	Shoots infected	Leaves inoculated	Leaves infected	Stems infected
	Number	*Per cent*	*Number*	*Per cent*	*Per cent*	*Number*	*Per cent*	*Number*	*Per cent*	*Per cent*
Vitis vinifera										
Inoculated	7	100	45	73.3	57.1	5	100	62	43.6	40
Control	2	0	13	0	0	3	0	25	0	0
Vitis labrusca										
Inoculated	10	100	40	47.5	20.0	5	0	28	0	0
Control	6	0	28	0	0	3	0	17	0	0
Vitis bourquina										
Inoculated	11	72.7	58	25.9	36.3	5	0	35	0	0
Control	4	0	31	0	0	2	0	18	0	0
Vitis rotundifolia										
Inoculated	11	0	112	0	0	8	100	80	26.3	100
Control	7	0	66	0	0	8	0	61	0	0

producing any external symptoms so long as the tissue was immature. As the leaf tissue approached maturity the extension of the fungus ceased, and the invaded tissue died and appeared as a spot in the leaf. The spots, therefore, had attained almost their full dimensions when they first appeared and did not enlarge in the mature leaves. For this reason the spots on leaves that were young when inoculated were large. In leaves that were almost mature when inoculated the fungus spread little, and, consequently, the spots were small. Since, however, the older leaves presented a greater surface area to the inoculum, the spots on these leaves were numerous. Apparently petiole tissue remains susceptible longer than leaf-blade tissue. Petiole cankers appeared on the first to the eighth leaf back of the growing tip of the shoot and on leaves as much as six cm. across at the time of inoculation. Severe petiole cankers sometimes resulted in the death of the entire leaf. Stem tissue was infected only from the first to the fifth nodes back of the growing tip. Occasionally the entire stem tip was blighted.

These results demonstrate that both *Guignardia bidwellii* and *G. bidwellii* f. *muscadinii* are capable of infecting leaves and stems of grapes and of producing typical black-rot lesions on them and that only immature host tissue is susceptible to infection by either organism. They show further that there is a difference in pathogenicity between *G. bidwellii* and its form. Of our native grapes *G. bidwellii* attacks only bunch grapes, while the form *muscadinii* attacks only muscadine grapes. Both fungi, however, are pathogenic to the European grape, producing similar symptoms on the leaves and stems. The fact that ascospores from perithecia on over-wintered leaves or berries produce cultures in which the pycnidial stage is formed and that pycnospores from these ascospore cultures produce black-rot lesions bearing typical pycnidia when inoculated on the leaves and stems of the host demonstrates the genetic connection of the perfect and imperfect stages of each of the two fungi.

CONTROL

In order to determine the protection against black rot afforded by spraying, experiments were conducted in an acre vineyard of the Hunt variety at the Georgia Experiment Station during the seasons 1942–1944. Half of the vineyard was not sprayed, while the other half was given four sprays each season. A 4–4–50 Bordeaux mixture was applied as follows: two sprays prior to blooming, one after the fruit was set, and the last when the fruit was half grown. Since there was some injury to young foliage, a 2–3–50 Bordeaux mixture was substituted for the first spray.

Spraying resulted in no increase in yields. The average yields per vine in the sprayed and in the unsprayed halves of the vineyard were as follows: 1942, sprayed—51 lb., unsprayed—69 lb.; 1943, sprayed—48 lb., unsprayed—48 lb.; 1944, sprayed—80 lb., unsprayed—84 lb. The average annual yields per vine for the three-year period were: sprayed—59.7 lb. (average of same vines for three years prior to test, 61 lb.), unsprayed—67 lb. (average of same vines for three years prior to test, 66 lb.).

In order to determine whether spraying reduced the amount of infection with black rot, even though it did not result in increased yields, counts were made in the vineyard immediately before harvest in 1943. Counts were made on five shoots on each of 20 vines, that is, on a total of 100 shoots in both the sprayed and the unsprayed halves of the vineyard. The results are given in table 3. The application of four sprays was not very effective in reducing the amount of black rot on the vegetative parts. It produced only a slight reduction in the percentage of leaves infected and in the percentage of stems cankered. More frequent spraying would probably result in a greater reduction. It is difficult to keep the foliage covered with spray, however, because of the rapid growth of the shoots. For example, the rate of growth of the Hunt variety was determined by tagging 25 shoots scattered throughout an acre vineyard and measuring the elongation of the stems and counting the number of new leaves formed at one- or two-week intervals during the 18 weeks from April 11 to August 16, 1945. The weekly rate of

growth was fairly uniform throughout this period except for the three weeks from April 19 to May 9 when unseasonable cold weather retarded growth. During the 18 weeks, the stems elongated an average of 2.98 in. per week and added an average of 2.07 new leaves per week. The most vigorous shoot averaged 6.88 in. stem elongation and produced an average of 3.78 new leaves per week. The highest rate of growth recorded for any shoot in a single week was a stem elongation of 10 in. with the addition of six new leaves. It is obvious that, even if the vines were sprayed at weekly intervals, a great amount of the most susceptible tissue would be exposed to infection shortly after each spray was applied. Apparently it would be impossible to obtain complete control of black rot by protective spraying in the presence of a continuous source of inoculum such as is furnished by unsprayed check rows.

TABLE 3.—*Effect of spraying on amount of black-rot infection on Hunt muscadine grapes at the Georgia Experiment Station in 1943*

Data recorded	Vines not sprayed	Vines sprayed	Reduction from spraying
			Per cent
No. of shoots	100	100	.....
No. of leaves	2271	2159	.....
Percentage of leaves infected and lost	52.58	49.33	6.18
Av. no. of spots per infected leaf	11.03	8.25	25.20
Percentage of stems cankered	74.00	66.00	10.81
Av. no. of cankers per infected stem	6.73	5.55	17.53
No. of fruit clusters	165	153	.....
Percentage of fruit clusters with pedicels infected	18.18	13.14	27.72
Av. no. of cankers per infected cluster	1.40	1.25	10.71
Number of berries	1300	1168	.....
Percentage of berries infected	30.70	16.01	47.85
Av. no. of cankers per infected berry	2.84	1.96	30.99

Of course, spraying an entire isolated vineyard in conjunction with the elimination of neighboring wild vines and cultivated vines which cannot be properly cared for, thorough pruning, and the disposal of all litter by burning during the winter would probably greatly reduce the amount of infection or even eliminate black rot in the vineyard.

Although only two of the four sprays were applied to the berries, these two sprays reduced the number of berries infected by approximately 50 per cent and also reduced the number of scabs and cankers on the infected berries by 30 per cent. It seems certain that more frequent spraying of the berries, perhaps at two-week intervals from the time the berries are set until they are full size, would give them adequate protection. Spraying the berries is difficult, however, because they are covered by a dense growth of stems and leaves.

DISCUSSION

Pathogenicity. The results obtained in the inoculation trials reported here are of interest because of the disparity in the results obtained by previous workers. Prior to 1900 several French pathologists reported studies in

which they apparently were able to demonstrate the pathogenicity of *Guignardia bidwellii* on bunch grapes as well as the genetic connection of the perfect and the imperfect stages of the fungus. Viala and Ravaz (10) inoculated berries with pycnospores and produced typical black-rot lesions on which pycnospores and later spermogonia formed. They also obtained infection of leaves inoculated with ascospores from overwintered berries, proving the pathogenicity of *G. bidwellii*, the genetic connection of the pycnidial and perithecial stages of the fungus, and the identity of the fungus on the leaves with that on the berries. Prunet (5) obtained infection of leaves by inoculating them with pycnospores. He observed the process of infection microscopically and found that the germ tubes penetrate the cuticle directly. He discovered that the incubation period depends upon weather conditions and that it varies from 22–25 days in April to 10–12 days in July. He also found that only young, growing leaves are susceptible. Mature leaves were not infected. Leaves that were not quite mature when inoculated but matured during the incubation period also were not infected. The germ tubes penetrated the cuticle of these leaves, but growth of the fungus within the host was arrested. The spots usually presented their full dimensions when first evident. Soursac (9) later attempted by means of inoculation trials to determine the relative resistance of various species of *Vitis*. He was able to obtain infection in the greenhouse only on *V. vinifera*, but his field inoculations were successful on a number of species of *Vitis*. Although these results seem to be conclusive, Reddick (6) reported that in experiments in New York he was unable to obtain proof of the pathogenicity of *G. bidwellii* on bunch grapes although he made thousands of inoculations both indoors and in the field. The results of my inoculation trials prove again the pathogenicity and connection of stages of *G. bidwellii* on bunch grapes and are in complete accord with those of Viala and Ravaz, Prunet, and Soursac. They demonstrate further the pathogenicity and genetic connection of the perfect and imperfect stages of *G. bidwellii* f. *muscadinii* on muscadine grapes. It is difficult to explain Reddick's failure to obtain infection with *G. bidwellii* unless his inoculations were made on mature tissue.

Forms of Guignardia bidwellii. The fact that the fungus associated with black rot of muscadine grapes differs in pathogenicity from *Guignardia bidwellii*, cause of black rot of bunch grapes, is of practical importance for the following reason. Since *G. bidwellii* f. *muscadinii* does not infect bunch grapes, it should be possible to plant native bunch grapes in the vicinity of muscadine grapes without danger of the disease spreading from the muscadine grapes to the bunch grapes. It should, therefore, be unnecessary to make costly attempts to control black rot on the muscadine grapes in order to remove a source of inoculum for the infection of the bunch grapes. On the other hand, since *G. bidwellii* f. *muscadinii* will readily infect *Vitis vinifera*, diseased muscadine vines would be a constant threat to plantings of European grapes in their vicinity.

The existence of a distinct form of *Guignardia bidwellii* explains Soursac's failure to obtain infection on *Vitis rotundifolia*. Soursac (9), using

inoculum of *G. bidwellii* from a single source, obtained infection on the following species which are listed in order of increasing resistance: *Vitis vinifera, V. arizonica* Engelm., *V. californica* Bentham, *V. labrusca, V. rubra* Michx., *V. monticola* Buckley, *V. coriacea* Shuttleworth, and *V. rupestris* Scheele. In another experiment he obtained infection also on *V. Lincecumii* Buckley, and *V. aestivalis* Michx. His inoculations produced no infection on *V. rotundifolia* because, as is now evident, *V. rotundifolia* is susceptible only to *G. bidwellii* f. *muscadinii.*

It seems probable that there are other forms of *Guignardia bidwellii* in addition to the form *muscadinii.* Soursac (9) failed to obtain infection on four American species of bunch grape, *Vitis riparia* Michx., *V. cordifolia* Lam., *V. cinerea* Engelm., and *V. candicans* Engelm., although black rot has been reported on all four of these species (8). This suggests the possibility that there are undescribed forms of *G. bidwellii* which attack only certain species of bunch grapes. Furthermore, black rot has been reported on Virginia Creeper (*Parthenocissus quinquefolia* (L.) Planch.) (1, p. 113R) and on Boston ivy (*P. tricuspidata* (Sieb. and Zucc.) Planch.) (2) and has been found on both of these species in Georgia. Preliminary inoculation trials indicate that the fungus associated with the disease on these two species, while it is morphologically similar to *G. bidwellii,* differs in pathogenicity from both *G. bidwellii* and *G. bidwellii* f. *muscadinii.* Further inoculation experiments will probably demonstrate that this is a distinct form of *G. bidwellii* which attacks only *Parthenocissus* species.

Importance of Black Rot on Muscadine Grapes. Although black rot is a common and conspicuous disease, it seems to have little direct effect upon yield of muscadine grapes. It is difficult to control; and control measures beyond good cultural practices (4, 7) such as selection of a suitable vineyard site, proper cultivation, fertilization, pruning, and vineyard sanitation would hardly be profitable. Upon the basis of present evidence spraying cannot be recommended for the control of black rot. With proper management good crops may be produced in the presence of this disease without any special measures being directed toward its control.

The only appreciable damage resulting from black rot is the lowering of the quality of the crop because of the presence of scabs on the berries. This is true of only a few varieties among which, however, is the most important commercial variety, the Hunt. This lowering of quality is of little importance at present because the crop is usually sold in bulk to wineries, and the dry, superficial scabs do not interfere with the use of diseased berries for the extraction of juice. If the crop should become important as a fresh or frozen fruit for table use, black-rot scabs would be objectionable.

Although there are varieties of good quality with resistant foliage and with berries that are almost immune, such as Dulcet and Thomas, these varieties do not yield as well as Hunt, a relatively susceptible variety. At present resistance to black rot is not, therefore, of primary importance in the selection of a variety. If, as seems improbable, black rot should become of major importance, it is well to know that there are resistant varieties of good

quality available. Furthermore, since resistance is present in some varieties, it should be possible to combine resistance with high yield and quality in future breeding work.

SUMMARY

Black rot, a common and conspicuous disease of muscadine grapes (*Vitis rotundifolia* Michx.), is primarily a disease of the vegetative parts. It causes a spot of the leaves, a canker or blight of the petioles, tendrils, stems, and flower clusters, and a superficial scab or canker of the berries. It causes little defoliation and only a negligible amount of berry drop. Black rot has little effect on yield and appears to be of minor importance.

Muscadine varieties vary considerably in degree of susceptibility to black rot. Some varieties, such as Dulcet and Thomas, have relatively resistant foliage and berries which are almost immune. These varieties, however, produce lower yields than Hunt, one of the most susceptible varieties.

The fungus associated with black rot on muscadine grapes differs in pathogenicity from *Guignardia bidwellii* (Ell.) Viala and Ravaz, cause of black rot of bunch grapes. Only slight morphological differences both in culture and in the field are associated with this difference in pathogenicity. The fungus is described, therefore, as a new form, *G. bidwellii* f. *muscadinii*. Inoculation trials have shown that of our native grapes *G. bidwellii* attacks only bunch grapes, while the form *muscadinii* attacks only muscadine grapes. Both, however, are capable of infecting the European grape, *Vitis vinifera*. Inoculations have also demonstrated the genetic connection of the pycnidial and perithecial stages of each of these fungi.

Experiments have produced no evidence that black rot on muscadine grapes can be profitably controlled by spraying. Control measures beyond good cultural practices necessary for the maintenance of vigorous growth are not recommended.

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LITERATURE CITED

1. BURGER, O. F. Report of Plant Pathologist. Ann. Rept. Florida Agric. Exp. Sta. 1924; 84R–113R. 1924.
2. EDSON, H. A. Black rot leaf spot of Boston ivy in the District of Columbia. U. S. Dept. Agr., Pl. Dis. Reptr. **21**: 251. 1937.
3. JENKINS, WILBERT A. Angular leaf spot of muscadines, caused by *Mycosphaerella angulata* n. sp. Phytopath. **32**: 71–80. 1942.
4. MURPHY, M. M., JR., T. A. PICKETT, and F. F. COWART. Muscadine grapes. Culture, varieties, and some properties of juices. Ga. Exp. Sta. Bull. 199. 1938.
5. PRUNET, A. Observations et expériences sur le black rot. III. Contributions a l'etude du parasité. Rev. Vit. **9**: 621–628. 1898.
6. REDDICK, DONALD. The black rot disease of grapes. New York (Cornell) Agric. Exp. Sta. Bull. 293: 289–364. 1911.
7. SAVAGE, E. F., *et al.* Further studies with the muscadine grape. Ga. Exp. Sta. Bull. 217. 1941.
8. SEYMOUR, ARTHUR BLISS. Host index of the Fungi of North America. Harvard Univ. Press, Cambridge, Mass. 732 pp. 1929.
9. SOURSAC, LOUIS. Recherches sur le black-rot. Ann. l'ecole nat. agr. Montpellier **8**: 151–160. 1908.
10. VIALA, PIERRE, and L. RAVAZ. Recherches experiméntales sur les maladies de la vigne. Compt. Rend. Acad. Sci. Paris **106**: 1711–1712. 1888.

STUDIES ON THE CAUSE OF STEM-END BROWNING IN GREEN MOUNTAIN POTATOES

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The term stem-end browning has been applied to a distinct type of internal discoloration in tubers of the Green Mountain (Fig. 1), Irish Cobbler, and a few other less common potato varieties. It is characterized by a marked necrosis of the xylem, phloem, and adjacent parenchyma cells of the stem end of the tuber (7). It rarely extends more than one-half inch beyond the stem scar. Very few diseased tubers are found at digging time but in some lots a high percentage of the tubers may develop the abnormality in storage. Maximum development is obtained by storing for about 100 days at 50° F. (4, 5, 6, 11).

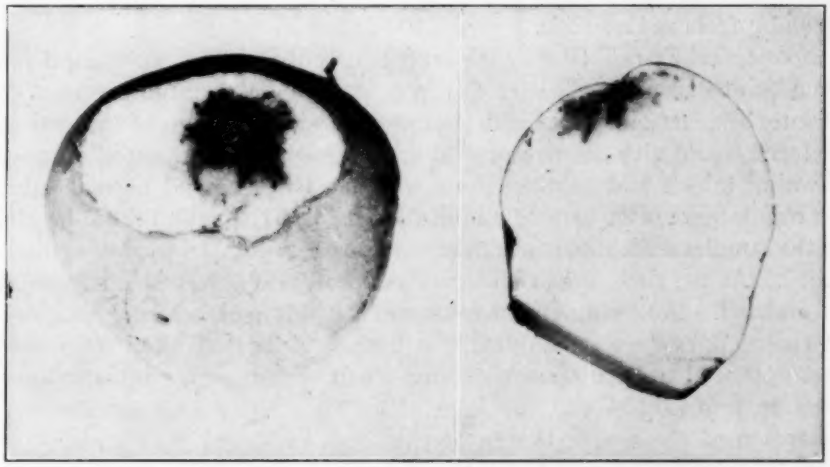

Fig. 1. Stem-end browning in Green Mountain tubers. (Photograph by M. T. Hilborn.)

Folsom and Rich (7) could find no evidence that the disease was caused by a virus. It has recently been shown, however, that in the case of Green Mountains the amount of stem-end browning in a crop is definitely related to the source of seed or, more specifically, to the commercial strain used (9). It was further demonstrated that in this variety there are two types of strains, differing in the tendency of tubers to develop stem-end browning. One type consistently produces a low percentage of tubers that develop the disease while two to seven times as many tubers of the other type may be diseased when grown and stored under identical conditions. Within a given strain, all tuber lines are equally predisposed to the disease. It is the purpose of this report to present data indicating that this difference in behavior

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among strains is apparently due to the presence of a virus, or virus strain, in those potato strains that normally produce a crop with a high incidence of stem-end browning.

The words "susceptible" and "resistant" as used herein are relative terms and are used to describe differences in tendency or predisposition to develop tuber symptoms referred to as stem-end browning. The term "Green Mountain strain" is used to differentiate between different commercial strains or growers' stocks. Although some of these are known by specific names, it does not necessarily follow that each differs genetically from the others.

EXPERIMENTAL WORK

The Green Mountain variety was used in all experiments. All field experiments were on Aroostook Farm, Presque Isle, Maine, or on an isolated seed farm in northern Maine. All tubers were stored at 50° F. for 100 days or longer before they were examined for stem-end browning.[2] The objective method described by Folsom and Rich (7) was used to differentiate stem-end browning from net necrosis.

Serological Tests. During the early stages of the work, serological technique as described by Chester (3) was used in an attempt to detect the presence of a virus in tubers with stem-end browning. Some of the anti-sera produced would give positive precipitin tests with most but not all stem-end browning tubers and negative tests with most but not all normal tubers. The results were often erratic, and at the time no explanation could be given for the samples with anomalous behavior, consequently the work was discontinued. At the time, the existence of resistant and susceptible strains was not realized. Following the discovery of the different behavior of strains, the earlier data were re-analyzed. It became clear that when the juice of what appeared to be a susceptible strain had been used for intraperitoneal injection into rabbits and the juice of an apparently resistant strain for absorption of the serum, the anti-serum thus produced gave positive precipitin reactions with potatoes selected from apparently susceptible strains and negative tests with those from strains that appeared to be resistant. In the latter case it did not matter whether or not the tuber had developed stem-end browning. In the former case, both normal and diseased tubers gave a positive reaction but the titer was much higher in the case of those showing stem-end browning. The data were interpreted therefore as indicating either the presence of a virus in the susceptible strains but not in the resistant ones or the presence of different strains of the same virus in the two types of Green Mountain potato.

Cage Experiments. As most potato viruses are spread by insects, a test was made of the effect of exclusion of insects on the incidence of stem-end browning. A field cage was constructed of aster cloth, half of which was entirely enclosed, and the other half left open at the sides to simulate the conditions inside the cage yet allow entry of insects. Both areas, as well as

[2] Acknowledgment is made to Michael Goven for making the examinations.

an adjoining unshaded and uncaged area, were planted with a resistant and a susceptible strain in alternate rows. To doubly insure the exclusion of insects, the caged plants were sprayed at intervals with nicotine sulphate. Half of the shaded rows and half of the unshaded rows were likewise sprayed with nicotine. The aphid population of the uncaged and unsprayed rows was rather high. The data in table 1 show that the tubers from the caged rows developed approximately the same amount of stem-end browning as did those from the others.

TABLE 1.—*Stem-end browning in Green Mountain tubers produced under a field cage*

Where grown in 1943	Insecticide spray, 1943	Stem-end browning			
		1943[a]		1944 (field planting)[b]	
		Minnesota lot	Keswick strain	Minnesota lot	Keswick strain
				Fields 1 and 2[c]	Field 2[d]
		Per cent	*Per cent*	*Per cent*	*Per cent*
Cage	Nicotine sulphate	4.5	34.4	9.3	40.3
Shaded area	Nicotine sulphate	6.2	31.3	.....	.....
	No spray	4.5	38.6	.....	.....
Open area	Nicotine sulphate	6.3	25.1	.....	.....
	No spray	4.7	30.1	13.0	39.3

[a] Figures based on 500 to 700 tubers.

[b] The 1943 crop was replanted in 1944 in an open field. Those grown outside the cage in 1943 were tuber indexed before planting. Tubers with leafroll, mosaic, etc., were discarded.

[c] Averages for 700 or more tubers from each field. Five single rows, each 29 ft. long, in Latin Square design, were used in Field 1; numerous small samples planted at random were used in Field 2. The figures for Field 2 were 9.5 per cent for the progeny of the cage-grown material and 13.8 per cent for the other sample.

[d] See footnote [c] for size of sample.

Samples were replanted the next year. In the case of the susceptible strain, the progeny of the cage-grown samples developed the same amount of stem-end browning as did that of the field-grown samples. In the case of the other lot, however, the progeny of the cage-grown samples produced less stem-end browning than did that of field-grown seed. The difference was not large and may not have been significant. The possible significance of the apparent increase will be discussed later. For the present the important thing to consider is that tubers of the resistant strain still developed much less stem-end browning than did those of the susceptible strain.

The Effect of Continued Propagation of Resistant Strains in Maine. The Green Mountain strain first recognized as being fairly resistant to stem-end browning was obtained from Minnesota. Consequently an experiment designed to test the effect of continued propagation of this strain was initiated. In 1942, this strain was obtained from a grower in Minnesota and propagated the following years either on a seed plot on Aroostook Farm or on an

isolated seed farm in northern Maine. These seed plots were rogued and harvested early to prevent the spread of leafroll and mosaic. Other lots of the strain were planted in ordinary fields and not harvested early but the crop was tuber indexed to eliminate leafroll and mosaic. In each of the following years, seed was obtained from the same Minnesota grower and compared with the locally propagated seed. Shortly after the recognition of the Minnesota lot as a resistant type, it was discovered that several resistant strains were prevalent in Maine. These strains have been grown in this State for many years. Hence, such strains were compared with the Minnesota lot, and the data are included in tables 2 and 3. While most of the seed used was harvested early, the test plots themselves were not.

TABLE 2.—*Stem-end browning in 1943 in Green Mountain potato strains propagated in Maine for various periods*

Field	Strain or lot	Years grown in Maine	Stem-end browning[a]
			Per cent
1[b]	Minnesota	First year	3.5
	Minnesota	First year	2.4
	Minnesota	Second year	6.7
	Keswick	Many	20.5
2[b]	Minnesota	First year	1.9
	Minnesota	Second year	5.8
	Keswick	Many	18.5
3[c]	Minnesota	First year	20.7
	Minnesota	Second year	22.4
	Highmoor	Many	19.5
	Keswick	Many	54.3
4[c]	Minnesota	First year	7.5
	Minnesota	Second year	9.1
	Highmoor	Many	11.3
	Keswick	Many	27.8
Average	Minnesota	First year	7.2
	Minnesota	Second year	11.0
	Keswick	Many	30.3

[a] After storage at 50° F. for 100 days or more.

[b] Each figure for this field is the average of 6 single row plots, each 29 feet long, Latin Square design.

[c] Each figure for this field is the average of 8 single row plots, each 29 feet long, Latin Square design.

No great change in susceptibility occurred in four years' time. Furthermore, the figures for the Vermont, Highmoor, and Phillips strains are about the same as those for the Minnesota lot with the possible exception of those for the year it was introduced. Since these strains have been grown in the State as long as or longer than the Keswick strain, it appears very probable that resistance to stem-end browning is a stable characteristic of those strains. The behavior of the Minnesota lot may be a little different. In most cases tubers of this lot had less stem-end browning the year of introduction than did subsequent crops. The differences obtained were usually small but they were consistent. This suggests that the tendency of this lot to produce

stem-end browning is increased slightly by propagation in Maine one or two years but that it does not increase after that. The level of susceptibility reached in that time appears to be identical with that of the Highmoor, Vermont, and Phillips strains. The data are insufficient to determine the cause of this change, although the data on the cage experiment (Table 1) suggest that an insect transmitted agent might be involved.

1943 Tuber Grafts. Eyeless plugs taken from tubers of the Keswick strain (No. 8 cork borer) were grafted into tubers of the Minnesota lot (holes cut with No. 7 cork borer). The holes were cut lengthwise of the tubers so that a longitudinal half could be used as a control. Twenty-seven of the grafted seed pieces together with the corresponding controls were planted

TABLE 3.—*Stem-end browning in Green Mountain potato strains propagated in Maine for various periods*

Year	Field	Strain	Years grown in Maine	Stem-end browning[a]
				Per cent
1944	5	Minnesota	First year	8.1
		Minnesota	Second year[b]	12.2
		Minnesota	Third year	15.2
	6	Minnesota	First year	8.7
		Highmoor	Many	6.9
		Vermont	Many	9.7
	7	Minnesota	First year	6.1
		Vermont	Many	7.5
		Phillips	Many	7.0
1945	8	Minnesota	First year	7.2
		Minnesota	Second year	10.7
		Minnesota	Fourth year	10.6
		Highmoor	Many	9.1
		Keswick	Many	31.6
	9	Minnesota	Fourth year	9.4
		Highmoor	Many	9.9
		Keswick	Many	36.6

[a] After storage at 50° F. for 100 days or more. Each figure is the average of 5 single row plots, each 29 feet long, Latin Square design.

[b] The seed used for this sample was not harvested early but it was tuber indexed. All other seed was harvested early.

under a field cage. Seven were lost because of seed piece rot or virus disease. In digging, the units were kept separate. The tubers produced by nine of the grafted units developed more stem-end browning than did those of the corresponding Minnesota control (Table 4). The amount of disease in the tubers from all grafted units was intermediate between that in the two controls, corresponding with what would be expected with a 40 per cent "take."[3] The nine units where a "take" was indicated gave fully as much stem-end browning as did the susceptible strain used for scions. These nine, as well as the corresponding controls were replanted in 1944. The tubers produced

[3] In view of other data presented, the assumption was made that in those cases where a "take" occurred, the stock was free of the virus causing or favoring stem-end browning. Where no "take" occurred, the presence of a mild strain of the virus was assumed.

by eight of these were dug and examined for stem-end browning. All of the replanted grafted units gave higher stem-end browning readings than did their corresponding Minnesota controls, but in three cases the increase was small. The figures for the other five grafted units were almost as high as were those for the Keswick strain.

Others of the tuber grafts made in 1943 were planted in an open field together with their corresponding controls. The units were not kept separate, hence the figures in table 5 are plot averages. The samples from the plots planted with the grafted seed pieces developed an amount of stem-end browning intermediate between that in samples from the two controls. One-third of the crop from each type of seed was replanted in 1944, and again the stem-end browning content of the progeny of the grafted seed pieces was

TABLE 4.—*Transmission by tuber grafts of a factor causing increased development of stem-end browning*

Data recorded	Keswick control	Grafted	Minnesota control
A. No. of units dug, 1943	20	20	20
B. Percentage stem-end browning, 1943	21.8	12.6	3.9
C. No. of units showing increase over Minnesota control, 1943	.....	9	.....
D. Percentage stem-end browning in C and corresponding controls	24.1	26.7	4.7
E. No. replanted C units and controls dug, 1944	8	8	8
F. Percentage stem-end browning in all units, 1944	40.3	29.8	8.3
G. No. replanted units showing some increase over Minnesota control, 1944	.....	8	.....
H. No. replanted units showing very definite increase over Minnesota control	.....	5	.....
I. Percentage stem-end browning in H and corresponding controls	43.3	36.9	11.5

intermediate. In both years, the crop from the grafted units had approximately the amount of stem-end browning that would be expected from a 25 per cent "take."[4]

1944 Tuber Grafts. About 100 new tuber grafts were made in 1944. The average figure on stem-end browning for the entire group was no greater than that for the Minnesota controls. Only ten of the grafted units produced tubers that developed as much stem-end browning as did those from the Keswick controls. The figures for 19 others were considerably higher than those for the Minnesota controls but less than those for the Keswick controls. Of these 29 units, 22 were free of net necrosis and were replanted in 1945, together with the corresponding controls. The progeny of only five of the grafted seed pieces contained an amount of stem-end browning similar to that in the progeny of the Keswick controls. These same five units were among those that had a high percentage of diseased tubers the previous year.

[4] Calculations from the 1943 data were made after making the assumptions outlined in footnote 3. All of the 1944 tubers were assumed to be infected with either a mild or a severe strain of the virus in question.

Hence, transmission of a factor favoring stem-end browning obtained in only about 5 per cent of the attempted grafts.

1944 Inarch Grafts. In 1944, transmission was attempted by means of inarch grafting. Tubers from the Minnesota lot were planted by tuber units (4 seed pieces per tuber). Two of the hills were left as controls and the other two used for grafting. Tubers of the Keswick strain were planted in 6-inch pots and the plants left in the greenhouse until used. When the field plants were 10 to 12 inches high, the Keswick plants were grafted to a stalk of the field-grown (Minnesota) plants by inarching. The field-grown hills were not trimmed to one stalk. Approximately 2 weeks later the stalk of the potted plant (Keswick) was severed below the graft. Several days later the plants were examined and only those where the Keswick scion remained green and turgid were marked for digging. In the year the grafts were

TABLE 5.—*Transmission by tuber grafts of a factor causing increased development of stem-end browning*

Type of seed used[a]	Stem-end browning	
	1943[b]	1944[c]
	Per cent	*Per cent*
A. Keswick strain	38.6	39.3
B. Minnesota lot	5.9	13.8
C. Eyeless plug of A grafted into a seed piece of B	15.4	21.1

[a] A and B seed consisted of unused portions of the tubers that were used for grafting.
[b] Average of 6 single row plots, each 29 feet long. Two Latin Squares.
[c] Average of 6 single row plots, each 29 feet long. Latin Square design. A representative portion of the 1943 crop was used for seed.

made, the grafted units produced tubers with three times as much stem-end browning as was found in tubers from the corresponding ungrafted units (Table 6). The figures on distribution of diseased tubers in the units indicate that there was a large increase in approximately one-third of the grafted units. In fact, about two-thirds of the grafted units showed some increase over their corresponding control, but in about half of these the increase was small.

Those units free of net necrosis were replanted in 1945 to see if the apparent increase was permanent. It appears that at least part of the increase was temporary, for the progeny of the grafted units produced only 1.4 times as much stem-end browning as the corresponding controls. This, in itself, may not mean too much for it has been shown that the relative amounts of stem-end browning in susceptible and resistant strains vary widely with environmental or seasonal factors (9). Of considerably more significance is the fact that many of the grafted units that had a high percentage of stem-end browning in 1944 also had a high amount in 1945, as shown by items H, I, and J in table 6. In item E, relative to replants from low stem-end browning units, the figure for the grafted units is but little larger than that for the controls, and the majority of the individual units

were low in stem-end browning (items F and G). The figure for replants of grafted units that had high stem-end browning in 1944 was considerably higher than that for the controls in 1945 (item H), and over 60 per cent of the units were high in stem-end browning (items I and J). There was little tendency for the control units which had high stem-end browning the first year to be high in stem-end browning in 1945. The 1944 and 1945 data for

TABLE 6.—*Transmission by inarch grafting of a factor causing increased development of stem-end browning*

Year	Item	Grafted[a]		Not grafted[b]	
		No.	Stem-end browning	No.	Stem-end browning
			Per cent		*Per cent*
1944[c]	A. All units	115	30.7	115	10.0
	B. Units with 0 to 25 per cent stem-end browning	60	.....	100	.....
	C. Units with over 25 per cent stem-end browning	55	.....	15	.....
1945[d]	D. All replants	60	18.8	42	13.1
	E. Replants of B units	24	15.9[e]	37	12.9
	F. B replants with 0 to 15 per cent stem-end browning	13	.....	25	.....
	G. B replants with over 15 per cent stem-end browning	11	.....	12	.....
	H. Replants of C units	36	21.1[f]	5	16.0
	I. C replants with 0 to 15 per cent stem-end browning	13	.....	4	.....
	J. C replants with over 15 per cent stem-end browning	23	.....	1	.....
	K. Total replants with 0 to 15 per cent stem-end browning	26	.....	29	.....
	L. Total replants with over 15 per cent stem-end browning	34	.....	13	.....

[a] Resistant strain used as stock and susceptible strain as scion.

[b] The figures in this column opposite items A and D are for the progeny of seed pieces from the same mother tubers as the grafted units. Those opposite items E and H are for tuber lines classified according to their behavior in 1944. In 1944 the 4 units from one tuber were planted in adjoining hills, and alternate hills were grafted. In 1945 the progeny of grafted and ungrafted units from the same mother tuber were planted in adjacent rows.

[c] Average of 6.3 tubers per unit.

[d] Average of 76.5 tubers per unit.

[e] The controls originating from the same mother tubers as the grafted units had 12.4 per cent stem-end browning.

[f] The controls originating from the same mother tubers as the grafted units had 15.9 per cent stem-end browning.

the grafted units gave a coefficient of correlation barely short of significance at the 5 per cent level. This fact does not necessarily indicate lack of correlation, for the sample size in 1944 was small (6.3 tubers per unit) and it has been found that with a given type of strain the individual hills may vary in stem-end browning content from zero to 100 per cent.

Since the grafted hills were not trimmed to one stalk, one would not necessarily expect the progeny of a successful graft to be fully as susceptible as the strain used as scion. This fact plus the fact that all units were not

replanted (because of leafroll infection) makes it difficult to estimate the percentage of successful transmission, but in general it appears that it would be of an order of magnitude of approximately 20 per cent. At least it is clear that in well over 50 per cent of the cases, no evidence of transmission could be detected.

1945 Inarch Grafts. In 1945 the same technique adopted the previous year was used except that both grafted and ungrafted units were trimmed to one stalk. The Highmoor strain, in addition to the Minnesota, was used as stocks. The results are presented in table 7. In the case of the Minnesota lot, grafting resulted in an increase in the average amount of stem-end browning and this was accompanied by a shift toward a larger number of hills with high stem-end browning content. With the Highmoor strain the increase in stem-end browning was small and the shift toward a higher number of high stem-end browning hills was likewise small. Until replantings are made the significance of these results is in doubt but it seems likely that,

TABLE 7.—*Transmission by inarch grafting of a factor causing increased development of stem-end browning. Experiments made in 1945*

Stock	Scion	Average number tubers per hill	Percentage of stem-end browning	Number of units having following percentages of stem-end browning		
				0 to 24	25 to 49	Over 50
Minnesota	Keswick	4.1	25.7	33	15	16
Minnesota	Not grafted	3.6	16.1	42	15	7
Highmoor	Keswick	3.8	25.5	35	26	15
Highmoor	Not grafted	3.4	20.0	43	20	13

at least in the case of the Minnesota seed, some transmission of a factor favoring stem-end browning was accomplished.

Tuber Inoculation. Several attempts were made to transmit the susceptibility factor by inoculating seed pieces of the Minnesota lot. Several methods were tried, including transmission by the cutting knife, by rubbing the two types of seed pieces together, and by dipping the seed piece into the ground pulp of stem-end browning tubers of the Keswick strain followed by bruising of the cut surface by a dull knife. In no case did the progeny of the inoculated seed pieces develop more stem-end browning than did those of the noninoculated pieces. The tubers were replanted the following year but no evidence of transmission was obtained.

Leaf Inoculation. In 1943, cage-grown plants of the Minnesota lot were inoculated with juice of Keswick plants by the leaf mutilation method using carborundum. Only 8.4 per cent of the tubers produced by the inoculated plants developed stem-end browning while 4.3 per cent of those from noninoculated controls had the disease. This difference was of doubtful significance as the number of tubers was only about 150; consequently the entire crop was replanted in 1944 in well-replicated plots. Ten per cent of the progeny of the tubers from inoculated plants developed stem-end browning

as did 9.9 per cent of that from the controls, indicating no transmission of the factor increasing the amount of stem-end browning. Leaf inoculation to many other species failed to establish the presence of any virus other than latent virus in the Keswick strain.

Stem-end Browning in Tubers Free of Latent Mosaic Virus (Virus X). A Green Mountain seedling was obtained from Dr. E. S. Schultz and examined for stem-end browning. Three tubers were found that had what appeared to be typical stem-end browning. Unfortunately there is no specific test for this disease, but the symptoms in these tubers were indistinguishable from stem-end browning in Green Mountain tubers. These tubers were tested for latent virus both by inoculation to peppers and to *Datura* and by serological methods. Portions of the tubers were planted in the greenhouse, and the plants so produced were tested for the presence of latent mosaic virus (virus X) both by inoculation and by serological methods. In all cases the results were negative. Parallel tests with Green Mountains that contained latent mosaic virus were uniformly positive. Consequently it was concluded that stem-end browning can occur in the absence of latent virus and, hence, that this virus is not the cause of the disease. Further evidence in support of this conclusion is the fact that the symptomatology on Turkish tobacco and *Datura* plants of the latent mosaic virus obtained from the Minnesota lot was indistinguishable from that of the latent mosaic virus from the Keswick strain.

Other Characteristics of Resistant and Susceptible Strains. Under some growing conditions or at least on some fields in some seasons, the resistant strains, especially that from Minnesota, have certain growth characteristics that distinguish them from the susceptible or Keswick strain. The former are sometimes taller, more erect, slightly lighter green; and their upper leaves are slightly more cupped. Their appearance resembles, in general, that of giant hill Green Mountains. These characteristics are not always apparent and in some seasons it is not possible to distinguish between the two types. That the resistant strains do not contain giant hill is indicated by the fact that on the average they produce more tubers per unit yield than the Keswick strain. No other difference has been noted. The two types of strains mature at about the same time and on the average yield equally well. Both are equally susceptible to leafroll infection and the development of net necrosis.

DISCUSSION

From the data presented in this report it is suggested that the Keswick strain of Green Mountain potato contains a virus or virus strain not present in the strains less subject to stem-end browning. The data are not sufficient to determine whether or not a virus is the actual cause of the disease. If such is the case, then it would be necessary to postulate the presence of different strains of the same virus in the two types of stocks. There are several facts that strongly suggest that such is the case. In all of the inarch grafts reported, organic union between stock and scion took place, for after removal

of the root and lower stem of the plant used as scion, the portion above the graft remained green and succulent. Once organic union is established, virus transmission readily takes place in the case of most viruses. If a plant contains one strain of a virus it is usually immune from another strain of the same virus. If the transmission is attempted by grafting, however, protection may be incomplete (1, 8). Hence, the results obtained are consistent with the idea that some of the Minnesota tubers were free of the virus in question, that the remaining ones were infected with a mild strain of the virus, and that the Keswick strain of Green Mountains carried a virulent strain of the virus. The other data support this view. There was a small but consistent tendency for the Minnesota lot to increase in proclivity to stem-end browning development the first year after introduction into Maine. On the average, this increase was of the order of magnitude of 30 to 50 per cent (Tables 2 and 3), suggesting that this percentage of the tubers were virus free when introduced but became infected during the first season in Maine. These figures agree very well with the percentage of grafted units that gave evidence of the transmission of an agent causing increased development of stem-end browning. That the Minnesota lot was not entirely free of the causal agent is indicated by the presence of some stem-end browning in the crop produced under a cage. That it was partially infected is indicated by the fact that the field-grown crop was 40 per cent higher in diseased tubers than was the cage-grown crop. The fact that the 30 to 50 per cent increase in stem-end browning usually noted after one season in Maine was delayed a second season where the crop was produced under a cage is of particular interest. A plausible explanation is that this increase is due to the transmission of a mild strain of the virus to 30 to 50 per cent of the units and that such transmission is prevented by exclusion of insects. The failure of the more virulent virus strain to spread to newly-introduced, virus-free potatoes is not necessarily in disagreement, for there are known cases where different strains of the same virus have different specific insect vectors (2, 10).

SUMMARY

A Green Mountain potato strain that normally produces a crop in which only a few tubers are affected with stem-end browning was made much more subject to development of the disease by tuber grafting and by inarch grafting. Tubers or plants of another strain, the tubers of which are highly predisposed to the disease, were used as scion material. The increased tendency toward stem-end-browning development was apparent not only the year the grafts were made but also in the progeny of the tubers produced by the grafted plants or seed pieces. Transmission was not obtained in all cases where organic union of the grafted plants occurred, nor was it obtained by mechanical inoculation methods. The tubers of a lot from another state became slightly more prone to the development of stem-end browning during the first year or two in Maine under field conditions but not when cage-grown. Otherwise the two types of Green Mountain strains are stable in

that there is no change from year to year in the relative amounts of stem-end browning developed in the crops of each.

The data are interpreted as indicative of the presence of a virus or virus strain in those lots highly subject to the disease and the absence of such a virus or virus strain in the other type. The hypothesis is advanced that a virus is the cause of stem-end browning and that different strains of the virus account for the difference in potentiality for stem-end-browning development evident in different commercial strains of the Green Mountain potato variety.

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LITERATURE CITED

1. BAWDEN, F. C. Studies on a virus causing foliar necrosis of the potato. Proc. Roy. Soc. (B) **116**: 375–395. 1934.
2. BLACK, L. M. Specific transmission of varieties of potato yellow-dwarf virus by related insects. Amer. Potato Jour. **18**: 231–233. 1941.
3. CHESTER, K. STARR. A critique of plant serology. I. The nature and utilization of phytoserological procedures. Quar. Rev. Biol. **12**: 19–46. 1937.
4. FOLSOM, DONALD. Temperature inhibition of storage development of net necrosis and "stem-end browning" of Maine potatoes of the Green Mountain variety. (Abstr.) Phytopath. **34**: 999. 1944.
5. ————, and MICHAEL GOVEN. Storage temperatures and stem-end browning. Maine Agr. Exp. Sta. Bull. 420: 439–440. 1943.
6. ————, and ————. Stem-end browning. Storage temperatures. Maine Agr. Exp. Sta. Bull. 426: 231–232. 1944.
7. ————, and AVERY E. RICH. Potato tuber net-necrosis and stem-end browning studies in Maine. Phytopath. **30**: 313–322. 1940.
8. KÖHLER, E. Mischinfektionen mit verschiedenen Stämmen des Ringmosaikvirus (X-Virus-Gruppe) der Kartoffel. (Untersuchungen über die Viruskrankheiten der Kartoffel. IV. Mitteilung) Angew. Bot. **17**: 60–74. 1935.
9. ROSS, A. FRANK. Susceptibility of Green Mountain and Irish Cobbler commercial strains to stem-end browning. Amer. Potato Jour. **23**: 219–234. 1946.
10. SEVERIN, HENRY H. P. Transmission of California aster and celery-yellows virus by three species of leafhoppers. Hilgardia **8**: 339–361. 1934.
11. UPTON, FRANK, and DONALD FOLSOM. Stem-end browning and net necrosis in storage. Maine Agr. Exp. Sta. Bull. 405: 502–503. 1941.

HEMP SEED TREATMENTS IN RELATION TO DIFFERENT DOSAGES AND CONDITIONS OF STORAGE

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Striking benefits in stand from the treatment of hemp seed were reported by Robinson.[2] This was a cooperative enterprise participated in by four state experiment stations and the U. S. Department of Agriculture, and was stimulated by the war needs for hemp. It seemed desirable that more be known about the efficacy of the more promising disinfectants, most effective rate of application, and the result when treated seed is stored for some time before planting. Although war needs are past, hemp has been and no doubt will continue to be grown in peace time. Furthermore, the results are of interest with regard to the treatment of seed in general.

MATERIALS AND METHODS

Hemp seed, *Cannabis sativa*, was obtained from Kentucky through the Hemp Division of the Commodity Credit Corporation, U. S. Department of Agriculture. A shipment of seed of the 1942 crop had a moisture content of 9.4 per cent which is unusually high for hemp. The germination on the seed information tag was given as 90 per cent. The seed was thoroughly mixed, and as hemp seed becomes very dusty from handling, the seed was recleaned with a fanning mill. Part of the seed was adjusted to a higher moisture, and the treatments were made a week later. Germination tests of the same seed lots were made 1 day, 3 months, and 7 months after treating with the disinfectants and dosages at the temperatures and moisture contents given in table 1. Another set containing 14.3 per cent moisture was stored at 70° F., but this became badly molded and was dead after 3 months, and therefore, those data are omitted. Seed of the 1943 crop from the same source had a moisture content of 7.6 per cent and was rated as 90 per cent germination on the seed information tag. Again part of the seed was modulated to a higher moisture content, and germination of the treated seed was tested after 1 day, 3 months, and 7 months storage at the temperatures and moisture contents given in table 2.

The moisture content of part of the seed was raised by adding water to the seed in a container that could be sealed, and then mixing well by shaking it once every day for a week. Treatments were made by tumbling a pint of seed with the disinfectant in a quart jar by means of a machine. Three commercial seed disinfectants were used: Arasan (50 per cent tetramethyl thiuramdisulphide), New Improved Ceresan (5 per cent ethyl mercury phosphate), and Spergon (98 per cent tetrachloro parabenzoquinone). Each one was used at 2 or 3 dosages, as shown in tables 1 and 2. After treatment, the

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[2] Robinson, Brittain B. Greenhouse seed treatment studies on hemp. Jour. Amer. Soc. Agron. 35: 911–914. 1943.

seed was stored in sealed pint jars filled nearly full. In this manner it was possible to keep the moisture constant. A recheck of the moisture content after 7 months' storage showed that the moistures were about the same as at the beginning except in the case of the moldy seed, in which moisture had risen from 14.3 to 14.6 per cent.

TABLE 1.—*Germination of hemp seed of two moisture contents after treating with three seed disinfectants and storing at 32° and 70° F. Experiment 1, Urbana, Illinois, 1943*

Storage temperature	Grain moisture	Seed disinfectant used for treating	Rate of application	Time of planting after treatment		
				1 day	3 mo.	7 mo.
	Pct.		*Oz./Bu.*	*Pct.*	*Pct.*	*Pct.*
32° F.	9.4	None	.....	57.0	49.3	29.5
		Arasan	0.5	69.6	64.0	47.3
		Do	1	68.0	64.3	50.8
		Do	2	70.0	60.0	55.0
		N.I. Ceresan	0.5	73.3	57.5	52.3
		Do	1	76.5	65.8	63.0
		Do	2	78.6	70.5	63.0
		Spergon	1.5	73.6	60.0	50.3
		Do	3	72.7	65.8	60.5
Least Significant Difference (5 per cent level)				10.5	9.0	12.1
32° F.	14.3	None	.....	55.8	45.3	33.3
		Arasan	0.5	65.7	60.8	32.0
		Do	1	72.0	60.5	44.0
		Do	2	73.5	61.5	47.8
		N.I. Ceresan	0.5	72.7	74.5	70.8
		Do	1	77.3	53.8	43.3
		Do	2	77.2	49.5	36.0
		Spergon	1.5	68.3	49.8	52.3
		Do	3	69.4	52.0	49.8
Least Significant Difference				9.7	10.6	9.9
70° F.	9.4	None	.....	56.3	43.8	9.9
		Arasan	0.5	67.9	58.8	24.3
		Do	1	69.5	53.0	24.0
		Do	2	71.1	57.3	27.8
		N.I. Ceresan	0.5	69.0	52.8	37.8
		Do	1	78.4	57.8	48.8
		Do	2	75.2	57.0	51.8
		Spergon	1.5	70.9	45.5	33.0
		Do	3	73.1	51.5	34.3
Least Significant Difference				12.7	10.3	9.4

The tests were made in greenhouse benches filled with dark colored soil brought from the University Farm. The greenhouse was kept at 65° to 70° F., as far as possible, but occasionally the temperatures went higher. The experiments were timed so that no tests were made during the hot summer months. Each test consisted of 4 to 6 randomized replications of 100 kernels for each item.

TABLE 2.—*Germination of hemp seed of two moisture contents after treating with three seed disinfectants and storing at 70° and 90° F. Experiment 2, Urbana, Illinois, 1944*

Storage temperature	Grain moisture	Seed disinfectant used for treating	Rate of application	Time of planting after treatment		
				1 day	3 mo.	7 mo.
	Pct.		*Oz./Bu.*	*Pct.*	*Pct.*	*Pct.*
70° F.	7.6	None	.....	72.7	69.8	65.7
		Arasan	0.5	91.0	89.0	85.3
		Do	1	93.3	90.0	88.2
		Do	2	92.0	90.5	89.7
		N.I. Ceresan	0.5	93.3	88.5	86.3
		Do	1	92.2	91.0	88.5
		Do	2	94.5	90.3	86.8
		Spergon	1.5	84.5	84.3	80.7
		Do	3	85.7	83.0	84.0
Least Significant Difference (5 per cent level)				7.5	9.1	9.5
70° F.	10.1	None	.....	67.0	61.0	47.2
		Arasan	0.5	81.8	77.5	59.8
		Do	1	86.0	79.0	64.0
		Do	2	90.2	79.3	61.0
		N.I. Ceresan	0.5	89.8	78.5	72.8
		Do	1	92.8	71.5	69.7
		Do	2	93.5	63.8	34.0
		Spergon	1.5	74.5	74.8	64.5
		Do	3	73.3	70.8	62.5
Least Significant Difference				10.3	9.2	9.5
90° F.	7.6	None	.....	72.7	65.3	50.5
		Arasan	0.5	91.0	86.5	63.8
		Do	1	93.3	85.8	67.0
		Do	2	92.0	87.5	73.8
		N.I. Ceresan	0.5	93.3	87.5	81.5
		Do	1	92.2	87.3	79.7
		Do	2	94.5	85.5	77.7
		Spergon	1.5	84.5	83.5	65.2
		Do	3	85.7	85.3	66.0
Least Significant Difference				7.5	6.8	10.5
90° F.	10.1	None	.....	67.0	43.5	0
		Arasan	0.5	81.8	55.3	0
		Do	1	86.0	52.0	0
		Do	2	90.2	59.5	0
		N.I. Ceresan	0.5	89.8	66.8	0
		Do	1	92.8	67.0	0
		Do	2	93.5	33.8	0
		Spergon	1.5	74.5	50.0	0
		Do	3	73.3	53.0	0
Least Significant Difference				10.3	12.4	.....

RESULTS

Striking increases in stand from treatment were obtained in both years with the three disinfectants used. This was due primarily to the preven-

tion of pre-emergence seedling blight. Some damping-off occurred also, but no significant differences were caused by seed treatment.

Seed Deterioration

The seed deteriorated very much in viability during 7 months' storage, especially that used in Experiment 1, which was low in vitality at the start. Seed deteriorated even when stored at 32° F. At this low temperature, the seed moisture, whether 9.4 or 14.3 per cent, made no apparent difference. In both cases the stand from the nontreated seed was reduced approximately half after 7 months' storage. When considering the average results from the use of the three seed disinfectants, at the most favorable dosage of each, the stand from treated seed was considerably higher than for the nontreated seed at the start; and after 7 months' storage the reduction in stand with treated seed was approximately 20 per cent.

When the seed was stored at 70° F. (Experiment 1), and the grain moisture was 9.4 per cent, deterioration in vitality was much more rapid than at 32° F. The reduction in stand in 7 months' storage time was 82 per cent when nontreated seed was used. In the case of treated seed, there was an average reduction of 48 per cent. When the grain moisture was 14.3 per cent, at 70° F. the grain was dead after 3 months and more or less moldy in spite of the treatments with disinfectants.

In Experiment 2, the grain moistures were lower, and one of the storage temperatures was higher than in Experiment 1. Initial germination was somewhat better, and deterioration in vitality was not so extreme in 7 months' time. In Experiment 2, which involved storage temperatures of 70° and 90° F. and grain moistures of 7.6 and 10.1 per cent, deterioration was least at the lowest temperature and moisture, and became greater with rise of either temperature or moisture. At the highest temperature and moisture, the seed germinated poorly after 3 months and was dead after 7 months' storage. Again, the lower the vitality of the seed, the more the stand was benefited by seed treatment.

Dosage

The only damage from too high a dosage occurred with New Improved Ceresan when used with seed at the higher moistures. This was true in Experiment 1 with grain moisture of 14.3 per cent, even though the storage temperature was only 32° F., and in Experiment 2 with grain moisture at 10.1 per cent and storage at 70° and 90° F. The injury was pronounced after 3 months' storage and worse after 7 months. The use of only ½ oz. per bushel of New Improved Ceresan caused no injury under any of the experimental conditions. However, with seed having only 7.6 and 9.4 per cent moisture, the higher rates, for the most part, gave the best protection.

Arasan used at ½, 1, and 2 oz. per bushel gave, in general, progressively better protection as the dosage was increased, irrespective of the temperature or grain moisture during storage. Spergon also had a tendency to give

better results when used at 3 oz. per bushel than when used at 1½ oz., irrespective of temperature or moisture.

If one keeps in mind the possibility of damage from a high dosage of New Improved Ceresan under some conditions, 1 oz. per bushel appears to be a good recommendation. Arasan was used to best advantage at 2 oz. per bushel. Spergon should probably be recommended at about 2 oz. per bushel. From the data in both experiments, New Improved Ceresan appeared to be most effective, Arasan second, and Spergon third. The differences, however, were not great and all of the treatments were beneficial.

DISCUSSION

The cost of hemp seed is much higher than that of the small grains, and the seeding rate in bushels per acre is about the same as for wheat. Therefore, the saving of seed, through obtaining better stands, is a very important

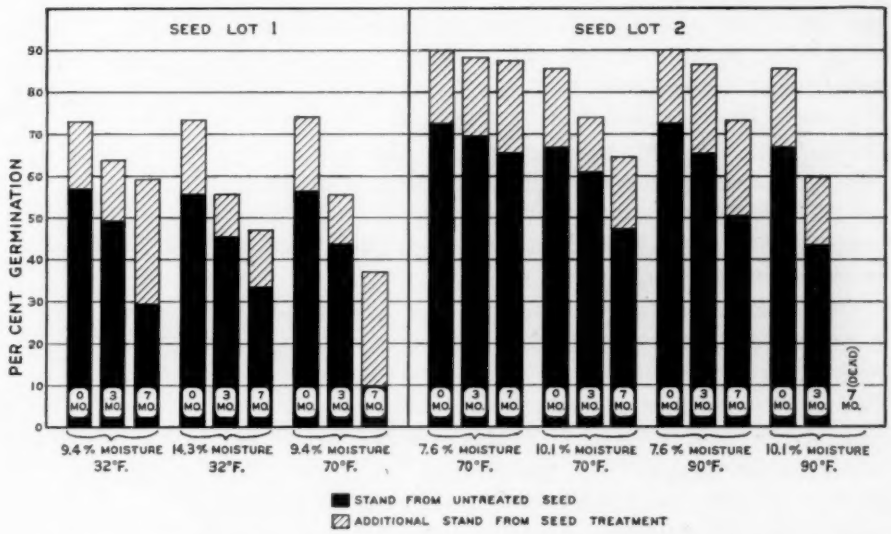

FIG. 1. Germination percentages of two lots of hemp seed when treated seed was held at several different moistures and temperatures for 0, 3, and 7 months before planting. Data are the averages for Arasan, New Improved Ceresan, and Spergon, used at the most favorable dosage.

consideration. If the 1¼ bushel-per-acre rate ordinarily used is satisfactory, then one bushel of treated seed should give at least as good stands according to these experiments. On the other hand, seed may be poor in vitality, unknown to the farmer, and if seed treatment is not used the poor stands obtained will result in coarse stalks which make a poor grade of fiber.

The black bars in figure 1 show graphically the drop in vitality as the seed ages. The planting after 3 months' storage was made in spring at approximately normal planting time, while the 7 months' storage lots were planted in September. None of the seed was stored long enough to be equivalent to seed carried over to the following year. The cross-hatched

part of the bars shows also that seed with poorest vitality benefited most from seed treatment. Therefore, seed caried over for an additional year would seem to be in special need of protection by a fungicide.

Of general interest for seed treatment knowledge of farm crops is the corroboration of existing fragmentary information that doses of Arasan, higher than ordinarily recommended, probably up to the amount that will stick to the seed appear to be harmless to seed vitality. With Ceresan, on the other hand, there is danger from an overdosage, and this is influenced to a considerable extent by the moisture content of the seed treated.

SUMMARY

Two seed lots of hemp were treated with Arasan and New Improved Ceresan at the rates of ½, 1, and 2 oz. per bushel, and with Spergon at 1½ and 3 oz. per bushel.

Grain moistures of 9.4 and 14.3 per cent and storage temperatures of 32° and 70° F. were used in 1943, and moistures of 7.6 and 10.1 per cent and storage temperatures of 70° and 90° F. were used in 1944. Storage periods from the time of treating until planting were 1 day, 3 months, and 7 months.

Seed vitality deteriorated rapidly in storage, even at 32° F. At 32° F. the differences in seed moisture had no apparent effect, but at higher temperatures, seed with the most moisture deteriorated most rapidly.

All three seed disinfectants produced striking increases in stand; the poorer the vitality of the seed, the greater the benefit, up to 400 per cent increase over the nontreated seed.

The maximum rate of application of Arasan and Spergon gave better average results than lower rates, and there was no indication of injury to seed vitality at any of the seed moistures or storage temperatures used. New Improved Ceresan, on the other hand, caused considerable injury when used at 2 oz. per bushel on seed that contained 10.1 or 14.3 per cent moisture and when such treated seed was stored for 3 or 7 months at 32° to 90° F. With grain moistures of 7.6 and 9.4 per cent, there was no apparent damage, and 2 oz. per bushel gave the best average stand.

From these experiments it would appear that for treating hemp seed, New Improved Ceresan should be recommended at the rate of about 1 oz. per bushel, while Arasan and Spergon should be used at about 2 oz. per bushel.

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BACTERIAL SPOT OF HONEYDEW MELON

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(Accepted for publication August 1, 1946)

Honeydew melons (*Cucumis melo* L. var. *inodorus* Naud.) bearing numerous water-soaked spots were first observed by G. B. Ramsey in a shipment from Colorado in September, 1940. Although cultures from these spots yielded bacteria in most instances, no attempts were made to identify the organisms.

On September 21, 1944, the Food Products Inspection Service at Chicago, Illinois, reported that in one carlot of honeydew melons from Colorado, 40 per cent bore sunken, water-soaked spots affecting from 10 to 75 per cent of the surface of the melons. Examination in the laboratory showed the diseased areas to be slightly sunken, circular to oblong, water-soaked, greenish-tan, and 2 to 6 mm. in diameter. On some melons the lesions had so coalesced that individual spots were indistinguishable (Fig. 1, A). Such areas were brown to black. Paraffin sections made from sunken, water-soaked lesions showed that the bacteria had entered the fruit through stomata and were between the parenchyma cells beneath (Fig. 1, B and C).[2]

Pure cultures were readily obtained by the dilution-plate and tissue-plant methods. Preliminary studies showed the organism to be motile by one to three polar flagella, rod-shaped with rounded ends, and Gram-negative. When it was grown in beef-extract agar and in asparagin agar, an abundant green-fluorescent pigment was produced. The same disease was again observed in August, 1945, on honeydew melons from Colorado and Arizona. Pure cultures obtained from these melons were identical in their biochemical and physiological reactions with those obtained in the 1944 isolations.

There is no mention in the literature of a similar disease of honeydew melon or even of the common muskmelon (*Cucumis melo* L.).

Carsner (4) in studies on the angular leaf spot of cucumbers was unable to obtain infection on 11 varieties of muskmelon from field or greenhouse inoculations with *Pseudomonas lachrymans* (E. F. Sm. and Bryan) Ferr. Anderson and Thornberry (2) report having observed a severe outbreak of angular leaf spot of cucurbits in Michigan in 1938 on seed cucumbers and a bacterial spot of pumpkin, squash, cantaloupe, and watermelon that closely resembled the angular leaf spot of cucumbers.

The physiological reactions of one of the white isolates (species undetermined) which Anderson and Thornberry obtained from cucumbers[3] that

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[2] Acknowledgment is made of the assistance of Barbara C. Heiberg who prepared the slides from which the photomicrographs were made.

[3] Data kindly supplied the writer by H. H. Thornberry.

were infected with a disease resembling angular leaf spot were compared with a number of isolates that the writer consistently obtained from diseased

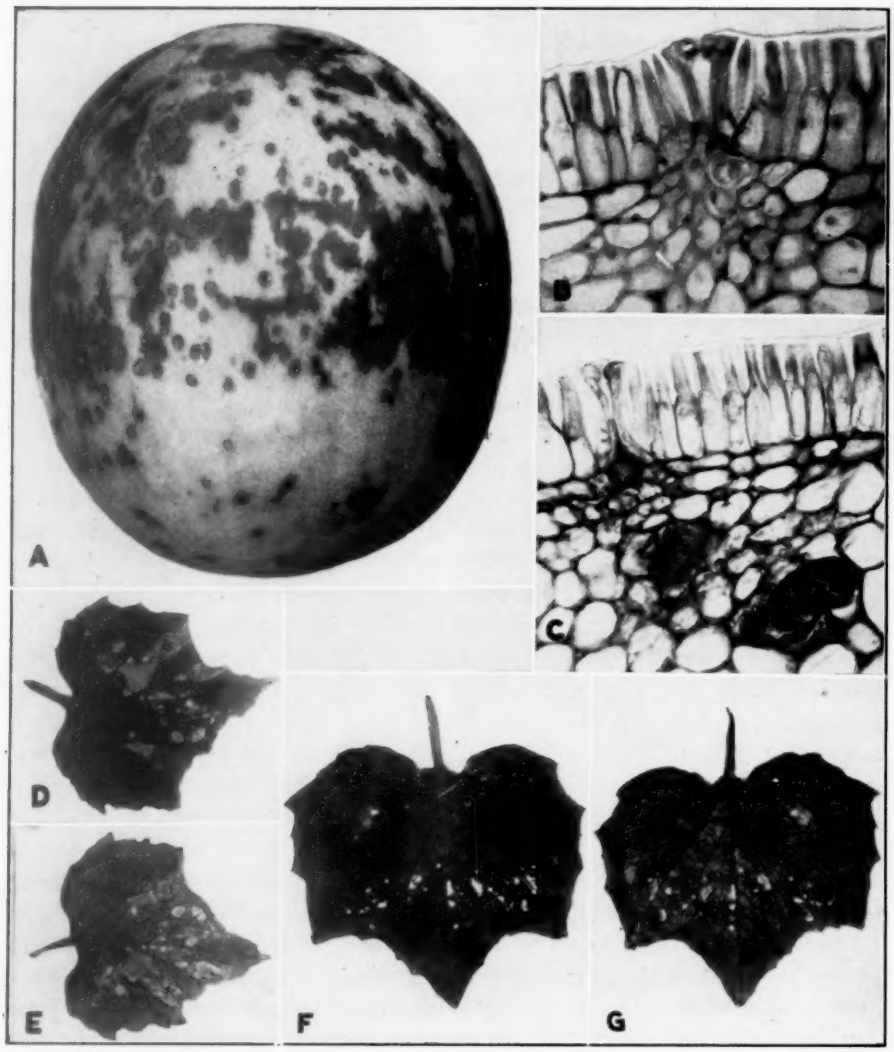

FIG. 1. A. Bacterial infection of honeydew melon. B. Cross section through epidermis of honeydew melon showing a normal stoma. 225×. C. Cross section through epidermis of diseased honeydew melon. Bacteria have entered through a stoma and are in tissues beneath. 210×. D. Infection of honeydew leaf by spray inoculation with honeydew isolate. E. Infection of cucumber leaf by spray inoculation with honeydew isolate. F. Infection of honeydew leaf 5 days after wound inoculation with *Pseudomonas lachrymans*. G. Infection of cucumber leaf 12 days after spray inoculation with *Pseudomonas lachrymans*.

honeydew melons. The results of the tests indicated that their isolate from cucumbers was distinct from the writer's melon isolates.

In the studies herein described, morphological and cultural character-

istics, biochemical reactions, and pathogenicity of a number of honeydew isolates were determined. The organisms were isolated in pure culture by the dilution plate method. Cultures were maintained in beef-extract agar (pH 6.8). Single colony isolations from cultures purified by the dilution-plate method were used in the studies. Six isolates from diseased honeydew melons were used in the morphological and biochemical studies. In all cases the tests were run in duplicate. Two authentic cultures of *Pseudomonas lachrymans* furnished by the American Type Culture Collection and four cultures of *Pseudomonas lachrymans* from cucumbers from a carlot shipment on the local market were included in the study for comparison.

MORPHOLOGY AND STAINING REACTIONS

Methods. For determination of form and size "negative" preparations from smears of the various honeydew and *Pseudomonas lachrymans* isolates from beef-extract agar were made with one per cent nigrosine. Fisher and Conn's (5) flagella stain was used to determine the number and position of flagella. Gram reaction was determined according to Hucker and Conn's (6) modification of Gram's stain. Ziehl-Nielsen's method was employed for acid fast properties. The Anthony (1) method of capsule staining was employed.

Morphological characters. The honeydew melon isolates are rods with rounded ends, occurring singly. Neither capsules nor spores were demonstrated. The cells measure 1.4 to 3.0 by 0.65 to 1.5 microns. They are readily stained by carbol fuchsin and crystal violet. The organisms are actively motile by one to three polar flagella, generally at one end. They are Gram-negative and not acid fast. In their morphological characteristics *Pseudomonas lachrymans* and the honeydew isolates are indistinguishable.

CULTURAL CHARACTERISTICS

Growth of the honeydew melon organisms and *Pseudomonas lachrymans* on beef-extract agar slants (pH 6.9) is white, flat, and butyrous, the medium becoming greened after three days. On beef-extract dilution plates after six days the colonies are white, slightly raised, circular, transparent, surface smooth, edges irregular. On potato-dextrose agar (pH 6.9) growth is slightly raised, filiform, white, and butyrous. Growth in nutrient broth (pH 6.9) is turbid after 24 hours, becoming greened after 48 hours. On asparagin medium (pH 6.8) growth after four days is white, flat, and butyrous. The medium is greened. Brom-cresol-purple milk becomes bluish purple and alkaline after 10 days, but no curd is formed. Growth of the honeydew organisms and of *Pseudomonas lachrymans* in plain gelatin stabs is evident in three days. Growth is first crateriform, later becoming stratiform. Liquefaction is complete in 21 days.

BIOCHEMICAL REACTIONS

Relation to free oxygen. When grown in Smith fermentation tubes growth is evident first in the open arms and later progresses to the domes indicating that the organism is a facultative anaerobe. There is no gas.

Nitrate reduction. The alpha-naphthylamine-sulfanilic acid test was employed on 1-, 2-, 5-, 7-, 10-, and 14-day-old cultures of the different isolates. The organism *Escherichia coli* was used as a control. Nitrate reduction did not occur in any of the honeydew or *Pseudomonas lachrymans* cultures.

Indole production. The various isolates growing in Bacto-tryptophane broth (pH 6.8) were tested by the Kovacs method (7) for indole production at the end of 7, 14, and 21 days. No positive reaction was obtained with any of the isolates. When *Escherichia coli* was used as a control organism a positive test for indole was obtained.

Ammonia production. Strips of filter paper saturated with a freshly prepared Nessler's solution were hung over 36- and 48-hour-old beef extract broth cultures of the various isolates. A positive test for ammonia was obtained.

Hydrogen sulphide. Strips of lead acetate paper failed to turn black when hung over beef-extract broth cultures of the various isolates, indicating that no hydrogen sulphide was being formed. *Escherichia coli* used as a control organism gave a positive test for hydrogen sulphide.

Hydrolysis of starch. Streak inoculations were made in beef-extract agar containing 0.2 per cent soluble starch. After five days, the surfaces of the plates were flooded with a saturated solution of iodine in 50 per cent alcohol. None showed any clear zone outside the area of growth indicating that no diastatic action had occurred.

The results of the cultural and biochemical tests with the honeydew organism and *Pseudomonas lachrymans* indicate that they are identical.

CARBON METABOLISM

Nine isolates from diseased honeydew melons and two isolates of *Pseudomonas lachrymans* were included in the studies of carbon metabolism. All tests were run in duplicate. Sugars, alcohols, and glucosides were added to the modified synthetic medium of Ayres, Rupp, and Johnson (3) as recommended in the Manual of Methods of Pure Culture Study of Bacteria issued by the Society of American Bacteriologists (8). The basal medium was adjusted to pH 7.0 and sterilized by autoclaving at 15 pounds pressure for 15 minutes. Brom cresol purple at a concentration of 0.02 per cent was added as an indicator. One per cent of some one of the sugars was added to the liquid basal medium before use for culturing. In order to avoid breaking down of the sugars during sterilization, lactose, levulose, maltose, raffinose, and sucrose were sterilized by filtration through a Berkefeld filter. Twenty-seven carbon sources were used. Inoculations were made in duplicate. Cultures were incubated at 27° C. Color change from purple to yellow was considered the indication of fermentation. Final readings were made at the end of 21 days. Organic acid media were prepared according to the Ayres-Rupp-Johnson method (3) except that 0.11 per cent acid was used. Brom thymol blue was used as an indicator. The criterion for fermentation was the presence of growth as indicated by turbidity and an increase in alkalinity.

The honeydew melon isolates and *Pseudomonas lachrymans* cultures utilized arabinose, xylose, sucrose, dextrose, levulose, galactose, mannose, and sorbitol. Rhamnose, maltose, lactose, trehalose, raffinose, melezitose, melibiose, cellibiose, starch, inulin, dextrin, glycogen, inositol, glycerol, dulcitol, erythritol, salicin, esculin, and arbutin were not fermented.

The honeydew melon isolates averaged more rapid fermentation of the carbohydrates than the *Pseudomonas lachrymans* cultures, but as has been stated, the final reactions of all the isolates were the same. The honeydew melon and *Pseudomonas lachrymans* isolates utilized malic, citric, and succinic acids but not acetic, lactic, formic, or tartaric acid. It appears from the foregoing that there are no significant differences in the carbon metabolism of the honeydew melon organism and *Pseudomonas lachrymans* and that they should be considered identical.

PATHOGENICITY

Three mature honeydew melons and 4 cucumber fruits were inoculated by needle puncture with 24-hour-old cultures from infected honeydew melons. Infection, as evidenced by water-soaked areas around needle punctures, was first observed on each of the honeydew melons 5 days after inoculation and on cucumbers 8 days after inoculation. After 11 days the infected areas on the honeydew melons were slightly sunken but there was no appreciable change in color. On cucumber, 11 days after inoculation, infected areas were slightly water-soaked and yellowish. Control melons and cucumbers inoculated by sterile needle punctures remained free of the disease. The organism was readily recovered in pure culture from the infected areas and these reisolates were used in further inoculation experiments.

In another experiment 2 mature honeydew melons and 4 mature cucumbers obtained on the local market were inoculated by spraying the uninjured surfaces with a water suspension of a 24-hour-old culture of one of the honeydew isolates. The cucumbers and melons were observed for 15 days. No infection was apparent during that time. A 24-hour-old culture of *Pseudomonas lachrymans* was used in spraying the uninjured surfaces of 2 honeydew melons and 4 mature cucumbers. No infection was apparent 15 days later. Uninjured melons and cucumbers inoculated with sterile water remained free of the disease.

Two mature honeydew melons and 3 cucumber fruits were inoculated by needle puncture with a 24-hour-old culture of *Pseudomonas lachrymans*. Infection of cucumbers was first observed 9 days after inoculation. After 15 days the infected area was very slightly sunken and light yellow. Infection of honeydew melons was noted 8 days after inoculation. After 15 days the infected area was sunken and yellowish-brown. Reisolations from infected honeydew melons and cucumbers were readily made. Control melons and cucumbers inoculated by sterile needle puncture remained free of the disease.

In greenhouse inoculations one series of 6 young honeydew melon and

6 cucumber plants growing in pots were sprayed with a suspension of a 24-hour-old culture of one of the honeydew isolates. A second series of 6 melons and 6 cucumber plants was sprayed with a suspension of a 24-hour-old culture of *Pseudomonas lachrymans*. After inoculation the plants were placed under bell jars for 24 hours after which they were returned to the greenhouse bench and held at 21° C. Infection was evident within 72 hours on foliage of the melon and cucumber plants inoculated with either the honeydew isolate or *Pseudomonas lachrymans*. Six days after inoculation the melon foliage inoculated with the honeydew isolate was so heavily infected that many lesions had coalesced (Fig. 1, D). Infection of cucumber leaves, inoculated with the honeydew organism or with *Pseudomonas lachrymans*, was moderate at the end of three days and showed considerable further spread at the end of six days though it was not so severe as on the melon plants. At this stage the lesions varied from mere dots to areas 1 to 2 mm. in diameter on each host when either the honeydew organism or *Pseudomonas lachrymans* was used (Fig. 1, F and G). Some of the lesions were more or less irregular and water-soaked. When dried, infected leaves were examined, the infected areas appeared chalky. The causal organisms were reisolated by plating from these lesions.

In another experiment 10 honeydew melon and 10 cucumber plants were used for wound inoculations. Four each of the melon and cucumber plants were inoculated by needle punctures into leaves, petioles, and stems with 24-hour-old agar slant cultures of *Pseudomonas lachrymans* and one of the honeydew isolates. Two of the melons and two of the cucumbers injured with a sterile needle, served as controls. Infection was evident within 48 hours on foliage of the melons and cucumbers inoculated by needle punctures with either the melon isolate or *Pseudomonas lachrymans* (Fig. 1, E). Petiole and stem infections were apparent 4 days after inoculation. Wilting was observed with both hosts but was much more rapid in the honeydew melon plants inoculated with the honeydew melon isolate. Melon and cucumber plants injured by sterile needle punctures remained free of the disease.

The foregoing inoculation tests show that the honeydew melon isolates and *Pseudomonas lachrymans* are pathogenic to fruits of honeydew melon and cucumber when needle puncture inoculations are made and to honeydew melon and cucumber plants inoculated by spraying with a bacterial suspension or with wounding. Because of the pathogenic similarity it is concluded that the melon organism should be considered identical with *Pseudomonas lachrymans*.

SUMMARY

A hitherto unreported bacterial disease of honeydew melons is described. A comparison of the morphological characteristics of the isolates from diseased honeydew melons with *Pseudomonas lachrymans* indicates that they are indistinguishable morphologically. The results of cultural and biochemical tests with the honeydew melon organism and *Pseudomonas lachry-*

mans indicate that they are identical. Studies have shown that there are no significant differences in carbon metabolism of the honeydew melon organism and *Pseudomonas lachrymans*. Pathogenicity studies show that both organisms are pathogenic to fruits, leaves, and stems of honeydew melon and cucumber. It is suggested, as a result of these studies, that the honeydew melon organism should be considered identical with *Pseudomonas lachrymans*.

U. S. DEPARTMENT OF AGRICULTURE,
CHICAGO, ILLINOIS.

LITERATURE CITED

1. ANTHONY, E. E. A note on capsule staining. Science **73**: 319. 1931.
2. ANDERSON, H. W., and H. H. THORNBERRY. Bacterial diseases in 1938. U. S. Dept. Agr., Pl. Dis. Rptr. **22**: 366–367. 1938.
3. AYRES, S. M., P. RUPP, and W. T. JOHNSON. A study of the alkali forming bacteria in milk. U. S. Dept. Agr. Bull. 782. 1919.
4. CARSNER, EUBANKS. Angular leafspot of cucumber: Dissemination, overwintering and control. Jour. Agr. Res. [U.S.] **15**: 201–220. 1918.
5. FISHER, P. J., and JEAN E. CONN. A flagella staining technique for soil bacteria. Stain Techn. **17**: 117–121. 1942.
6. HUCKER, G. J., and H. J. CONN. Further studies on the methods of Gram staining. N. Y. (Geneva) Agr. Exp. Sta., Tech. Bull. 128. 1927.
7. KOVACS, N. Eine vereinfachte Methode zum Nachweis der Indolbildung durch Bakterien. Zeitschr. f. Immunitats. **55**: 311–315. 1928.
8. SOCIETY OF AMERICAN BACTERIOLOGISTS. Committee on bacteriological technic. Manual of methods for pure culture study of bacteria. Leaflet 2. Ed. 9. p. 14. 1944.

A NEW RACE OF CERCOSPORA ORYZAE ON RICE[1]

S. J. P. CHILTON AND E. C. TULLIS[2]

(Accepted for publication August 24, 1946)

Five races and several subraces of *Cercospora oryzae* Miyake were reported by Ryker[3] in 1943 as occurring on rice in Louisiana. These races were differentiated by their differing range of pathogenicity on the following rice varieties: Blue Rose, Blue Rose 41, Fortuna, Caloro, Colusa, Zenith, Delitus, and Southern Red rice. Before 1944, the variety Rexoro had been resistant to all of these races both in commercial fields and in experimental plots artificially inoculated. In 1944, however, and again in 1945, Rexoro was susceptible both in Texas[4] and in Louisiana.[5, 6]

Texas Patna and Bluebonnet, two new rice varieties of which Rexoro was one of the parents, also were susceptible. In 1945, the disease was widespread on Rexoro throughout the rice area of Texas and Louisiana. The fungus was isolated from Rexoro and Texas Patna and used to inoculate differential varieties. The results substantiated the indications that the fungus on Rexoro and Texas Patna is a new race of *Cercospora oryzae*. It is the purpose of the present paper to report the disease reactions of the differential varieties used and to summarize the results of artificial inoculations with the new race on commercial varieties and varieties and selections introduced from foreign countries, so that the information may be available for use in breeding for resistance to this new race. This information is particularly important because Rexoro was the most important high-quality, long-grain variety of rice grown in the United States in 1945.

MATERIALS AND METHODS

From a number of cultures isolated from lesions on plants of Rexoro and Texas Patna, one culture from Texas Patna from Texas and one from Rexoro from Louisiana were used in June to July to inoculate young rice plants of 6 differential varieties and 24 commercial varieties growing in rod rows at the Rice Experiment Station, Crowley, La., and at Texas Substation No. 4, Beaumont, Texas. At the latter station, 703 foreign varieties and selections from various parts of the world also were similarly inoculated with the culture of the fungus isolated from Texas Patna.

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[3] Ryker, T. C. Physiologic specialization in *Cercospora oryzae*. Phytopath. **33**: 70–74. 1943.

[4] Tullis, E. C., and G. E. Altstatt. Rice diseases in Texas in 1944. U. S. Dept. Agr., Pl. Dis. Reptr. **28**: 1103–1104. 1944.

[5] Chilton, S. J. P. A new race of *Cercospora oryzae*. La. Agr. Expt. Sta. Ann. Rept. 1943–1944: 112. 1945.

[6] Chilton, S. J. P., and E. C. Tullis. A new race of *Cercospora oryzae* on rice. (Abstr.) Phytopath. **36**: 395. 1946.

The method of inoculation was the same as that described by Ryker[3] as his method No. 2. That is, spore suspensions from pure cultures were sprayed on the young rice plants in the evening between six o'clock and dark.

The ratings used to record the disease reaction, that is the susceptibility or resistance of the various varieties, were: S = susceptible, MR = moderately resistant, and R = resistant. The susceptibility or resistance of a variety was determined (1) by the length of time elapsed after the inoculation before the lesions began to appear, and (2) by the size of the lesions when fully developed. On susceptible varieties, lesions began to appear 10 to 12 days after inoculation and usually were 7–10 mm. long at the end of three weeks. The moderately resistant varieties reacted similarly except that the incubation period was two or three days longer and the lesions were shorter. On resistant varieties, lesions did not appear until about 18 days or more after inoculation, and they remained small.

REACTIONS OF DIFFERENTIAL VARIETIES

The disease reactions of the six differential varieties to the previously described races 1 through 5 and to the new race, designated as race 6, are given in table 1.

TABLE 1.—*Disease reaction of differential varieties to six races of Cercospora oryzae*

Variety	Reaction to					
	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6
Blue Rose	S	MR	S	R	R	R
Blue Rose 41	R	S	R	R	R	R
Caloro	R	R	S	R	R	R
Fortuna	R	R	R	S	R	R
Red Rice	R	R	R	R	S	....
Rexoro	R	R	R	R	R	S

Rexoro is resistant to races 1 through 5, and it is the only one of the differential varieties that is susceptible to the new race 6.

RELATIVE RESISTANCE OF VARIETIES

The commercial varieties of rice found to be resistant to race 6 include (1) the short-grain: Acadia, Asahi, Caloro, Cody, and Colusa; (2) the medium-grain: Arkrose, Blue Rose, Blue Rose 41, Calady 40, Early Prolific, Hill Medium, Kamrose, Magnolia, and Zenith; and (3) the long-grain: Arkansas Fortuna, Delrex, Fortuna, Lady Wright, Nira, Nira 43, and Prelude.

The commercial varieties susceptible to race 6 were all long-grain: Bluebonnet, Rexoro, and Texas Patna. Bluebonnet, Texas Patna, and a selection known as Nirex, which also was susceptible, were developed from crosses in which Rexoro was one of the parents.

Of the 703 foreign varieties and selections, 555 were resistant to race 6, while 109 were moderately resistant and 16 were susceptible. The other 23 appeared to be segregating. Among the 555 foreign varieties and selections resistant to race 6, were the following glutinous varieties, some of which may be found of value in California: Asahi mochi, Bozu mochi, Myogahari mochi, and Mochi Gomi.

DISCUSSION

The rice varieties Bluebonnet, Texas Patna, and Nirex, all susceptible to race 6, were developed from crosses in which Rexoro was one of the parents. Their susceptibility to this race clearly came from Rexoro, the female parent, as both Fortuna, the male parent of Bluebonnet, and Nira, the male parent of Nirex, are resistant to race 6. The reaction of the male parent of Texas Patna, C.I. 5094, to race 6 is not known, but presumably it is resistant. Likewise, the reaction of Delitus, which unfortunately was not included in the tests, is thought to be resistant, because Delrex, which was developed from a cross between Rexoro and Delitus, is resistant. That is, the Delitus parent undoubtedly carried the factor for resistance in Delrex. This is the more likely, because, as previously reported,[6] an F_2 generation in a cross between a resistant variety and Rexoro segregated in a ratio of 3 resistant to 1 susceptible.

SUMMARY

A new race of *Cercospora oryzae*, designated as race 6, is reported on rice in Louisiana and Texas. Race 6 attacks Rexoro and certain other commercial varieties of Rexoro parentage. The reactions of the differential varieties of rice used to separate the various races of *Cercospora oryzae* as well as the reactions of a number of other commercial varieties and foreign introductions are reported.

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BEAUMONT, TEXAS

THE PREPARATION OF MANUSCRIPTS FOR PHYTOPATHOLOGY[1]

A. J. Riker

WRITING—A PART OF RESEARCH

The publication of results from research is an important matter because of the permanence of the record and its use by others. The manner in which it is done reflects not only upon the individual worker, but also upon the organization of which he is a part. The preparation of a manuscript which accurately conveys readable ideas is an essential phase of research; it is just as valuable as doing more experiments; and it is fully as worthy of our best efforts (cf. Allen, 1). Here stands our brain child. How shall we dress it for presentation to the world?

Writing is one of our most important arts. It crystallizes for us the experiments and ideas of others, living and dead, near at hand and in a far country. It gives us the combined experience of the human mind to use and to pass on, with such additions as we ourselves can make.

Making these additions through printed papers sometimes causes both headaches and heartaches. Recognizing that not every member can ride Pegasus well, The American Phytopathological Society has requested a new (cf. Reddick, 18) series of recommendations on writing manuscripts. It has also cracked the whip over the Editorial Board in a mandate "to sharpen editorial pencils," to employ at least two qualified and anonymous referees on each manuscript, and to improve the quality of papers published through suggestion and encouragement, as far as possible, and to reject papers when necessary.

By this action the Society is not reaching for perfection but is striving to maintain a reasonably high technical standard, to avoid unnecessary burdens on the unpaid editorial staff, to meet the greatly increased cost of printing, and above all to prevent our science from sinking under the very bulk of present-day publication (cf. Weiss, 23). Although the subject is pathological, the report of it need not be pathological too.

Our purposes are to diagnose some of the diseases of manuscripts and, for those who want them, to suggest both prevention and cure.

A skillful investigator avoids rushing into print with an account of "flea" research. By this we mean the kind in which he jumps on this sub-

[1] This article was prepared at the suggestion of the Council of The American Phytopathological Society and at the invitation of the other members of the Editorial Board of Phytopathology. These and various other friends and colleagues, including particularly C. E. Allen, Noble Clark, R. E. Cleland, J. G. Dickson, H. B. Humphrey, G. W. Keitt, Ross Marvin, M. C. Merrill, and J. C. Walker, have given many valuable suggestions. Mesdames I. B. Hodgson and E. K. Marks have assisted with many details. Much of this material already has been written (19), and all of it has been collected freely wherever found. There is no intention whatever to say or to imply, "This is it; there is no other way." Rather the hope is to provide assistance for those who want it, and who desire some understanding of the reasons behind the specific directions given.

The illustrations were drawn by Eugene Herrling.

The manuscript was approved for publication by the Director of the Wisconsin Agricultural Experiment Station.

ject for a bite and on that subject for another. Nothing is really accomplished except possibly starting an irritation and a place to scratch.

The results of research are valid only in accord with the reliability of the methods employed and with the accuracy of their interpretation. After an observation has been recorded or an experiment has been made, a strict and seldom-broken rule requires that a report of such work must not be published for the use of others until repeated determinations have been made and before the results have been satisfactorily analyzed. The simpler experiments are commonly made at least in duplicate or triplicate with suitable controls and are carried through three separate times. A good investigator does not become so enthusiastic about an experiment that he fails to view it impartially and to accept sound evidence against it. On the contrary, before publishing he makes every reasonable effort to find an error in the procedure followed, in the experiment itself, or in the conclusions drawn from it.

The reproducible experiment is essential for scientific progress. So the writer should give the reader all information necessary for repeating the work. Likewise, the writer should be reasonably sure that a competent man, when he repeats the work, will find similar data and parallel conclusions.

However, when significant results are actually ready, nothing should delay a prompt and adequate release. Probably as many writers commit sins of omission, which are hard to see, as sins of commission. Scientists in publicly supported institutions have a special responsibility to give promptly to the public the benefit of their verified investigations. The first and authoritative announcement of new research is best made before a scientific society or through publication in a technical magazine. Simultaneous popular releases, when desirable, can be arranged for the press and radio. Such releases must have an entirely different style, which few technical men ever achieve. For such writing a journalist may be secured, providing the scientist corrects his technical errors.

Since different writers achieve satisfactory technical manuscripts by various means, the following suggestions apply only in case there is no better way. Some pains have been taken to explain the philosophy behind certain recommended procedures. These did not descend ready-made from "Minerva" in an armchair; they have developed through many years of toil; they have come from disappointment as well as from satisfaction.

FINE FIGURES MAKE FINE PAPERS

Writing is obviously much easier when one has the subject matter clearly in mind and actually before him. Consequently, many skillful investigators first prepare—at least in temporary form—their pictures, line drawings, graphs, and tables. These items, as well as the text, they condense as much as may be consistent with clarity.

Illustrations Are Never Too Good

The first impression an article makes commonly depends on the character of the illustration. A good piece of writing loses considerable value if the

illustrations are poor. To be sure, one may not be entirely just, but he is realistic to feel that fine figures make fine publications. The best possible text figures are never better than they need to be!

Illustrations are usually photographs or line drawings. As part of the printing procedure, these are made into engravings: the photographs are converted into half tones, the line drawings into zinc etchings.

Scientific journals cannot afford to use illustrations as mere embellishments for a paper. Each illustration must serve a purpose: it should explain a new technique or apparatus; it should amplify a taxonomic description; it should emphasize the appearance of experimental material; or it should summarize data that are too numerous to present in the text.

If he has several photographs, the author should group them together for economy wherever possible. The same applies to line drawings. Photographs and drawings, however, are seldom combined.

Photographs

First, let us consider photographs, which should be taken so that unimportant and distracting items are left out. A glossy finish gives best reproduction (**10, 11**). Prints should have clear, white backgrounds and strong contrasts, because the half tones usually have less contrast and less detail than the originals.

Photographs are mounted on heavy paperboard, one side of which has a hard, smooth, white surface. Although paste may be employed, wrinkles frequently appear that make the illustrations unacceptable. Rubber mounting cement is commonly used as follows: the back of the print and the smooth, white face of the paperboard are both smeared with a thin layer of the mounting cement. After drying a few minutes, when both appear sticky, the photograph is laid in position and pressed down with a cloth. Any excess of cement on the paperboard or picture is removed by rubbing with a cloth or soft eraser.

When several pictures are mounted together in one figure, each one is lettered, *e.g.*, A, B, C, D, . . . in the lower left-hand corner from left to right and from top to bottom. The waxy surface on all glossy prints is cleaned away from a small area with a touch of ether or an eraser, and the lettering is done with India ink directly on the picture. To do this the author employs either a skillful hand or a commercial lettering guide. If the picture is black, the letter may be outlined with white ink or placed on a small white ink circle. Pasted, cut-out, block letters usually are too heavy to look well and have a poor chance of sticking on the waxy surface.

Because the photographs or drawings must go to an engraver, they are separated from the manuscript and need complete identification. Each illustration desirably carries the following information: (1) the name of the author, (2) the title of the manuscript, (3) the figure number, and (4) the legend, here as well as with the text. These items may be neatly written or, preferably, typed on white paper, then pasted on the paperboard back.

Legends should appear in the text where the first reference is made to the figure. Although it should be brief, it should ordinarily make the illustration clear without reference to the text. Magnifications given for photomicrographs should be calculated according to the size as finally printed.

Finally, whether with a photograph, drawing, or graph, much tender care in preparing good figures can go down the drain unless they are well protected. Innocent paper clips or pencil marks on the previous page can make ruinous dents. Smudges from soiled fingers or carbon paper often show up surprisingly in printing. Protecting the picture with a "flap" of thin paper, pasted first on the paperboard back, then drawn over the face, is decidedly worth while. Likewise, pictures need heavy cardboard protection against creases, folds, and broken corners in the mail. How many writers learn this the hard way!

Drawings over Photographs

When a photograph or a photomicrograph is poor or contains much distracting, unnecessary material, which cannot be trimmed away, the author can make a line drawing on the print (13). Best results come from an enlargement done on paper with a matte surface. The drawing is then made with waterproof black ink directly over the wax-free photograph. After the ink has been thoroughly dried, the silver image of the print is removed in an iodine solution, made by grinding 5 gm. of iodine and 15 gm. of potassium iodide in a little water, until they are dissolved, and by diluting to 500 ml. with water. A few minutes after all traces of the photograph have disappeared, the print is rinsed in water and then the iodine color is bleached for about 5 minutes in 20 per cent "hypo" solution. Because the waterproof ink smears easily, it must not be touched while the paper is wet. The print is washed in water, pinned to a flat, blotting surface, dried in a vertical position, and pressed with a flatiron. Some further drawing often is necessary on the dried print.

Photographs were considered in earlier days to be more convincing than drawings because "photographs don't lie." This naive idea was long ago given up in recognition of photographic skill,[2] even without retouching. Whichever presents the evidence more clearly is obviously the one to use.

Originals Made to Fit the Page

The size of an original illustration, whether photograph, drawing, or graph, requires adjustment in relation to the printed page. An author can frequently trim from photographs much unnecessary surface, such as flowerpots, tops of test tubes, or background, and thus allow more space for the center of interest; or he turns some photographs sidewise for an improved arrangement. Graphs and drawings usually come out best when they are made 3 or 4 times as large as the final cut because small imperfections dis-

[2] Most fishermen know that if they should hold a 12-inch fish at arm's length in front of a yardstick and should photograph it with an ordinary camera, the film would record a fish over 18 inches long.

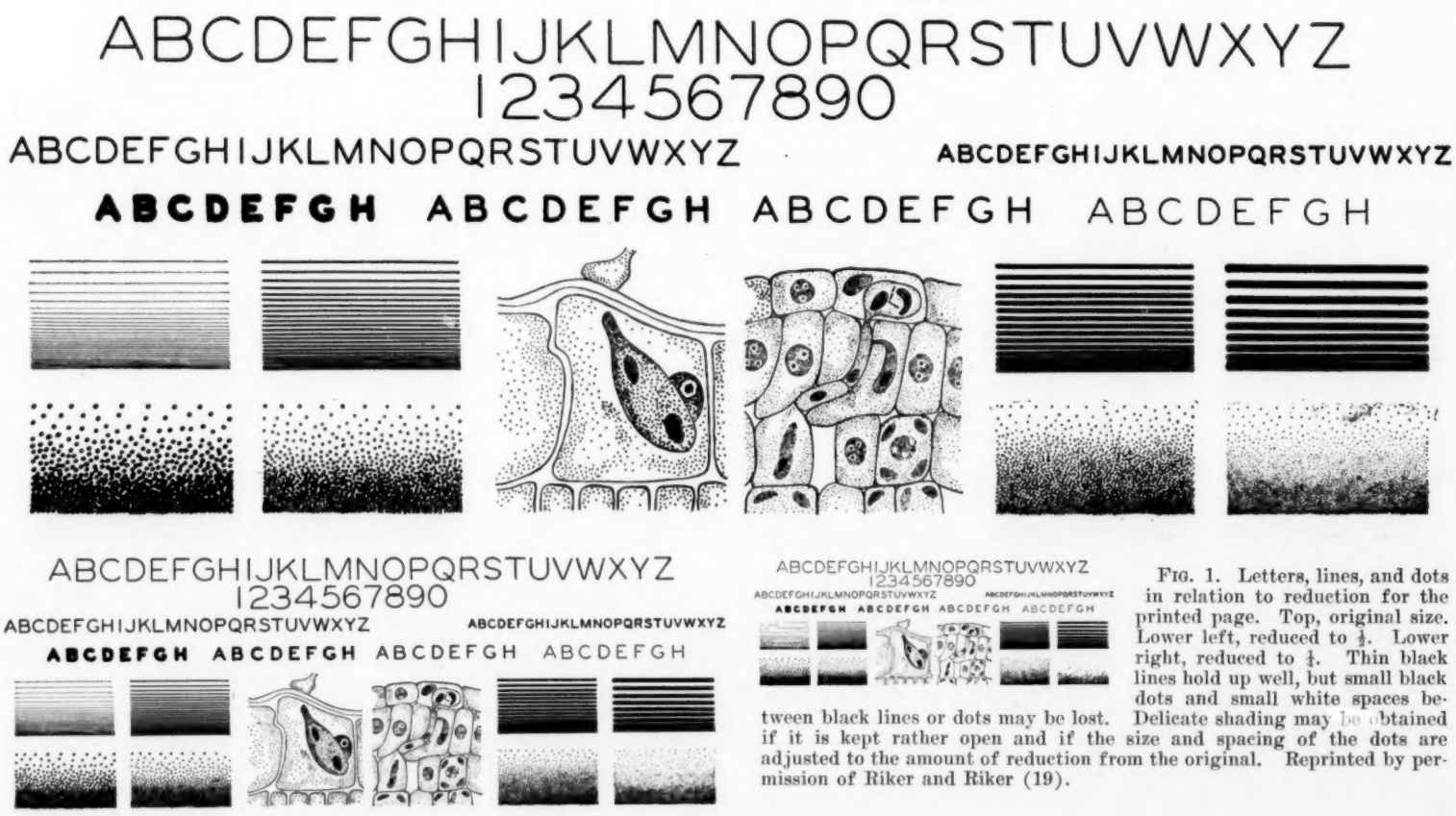

FIG. 1. Letters, lines, and dots in relation to reduction for the printed page. Top, original size. Lower left, reduced to ½. Lower right, reduced to ¼. Thin black lines hold up well, but small black dots and small white spaces between black lines or dots may be lost. Delicate shading may be obtained if it is kept rather open and if the size and spacing of the dots are adjusted to the amount of reduction from the original. Reprinted by permission of Riker and Riker (19).

appear with the reduction. The original illustration can be made with dimensions that fit the page[3] in the following way: On the bottom of a large sheet of paper the author draws a line on which are marked 1, 2, and 4 times the width of the printed page. At the left end of this base line he draws a vertical line on which he marks the height of the page, less the space the legend will occupy. This he approximates by counting the number of words in the legend and by measuring an already printed legend of similar length. He then marks on the vertical line 1, 2, and 4 times the corrected page height, and he completes the corresponding 3 rectangles. He constructs intermediate rectangles as desired. If the final illustration is to occupy the entire page, $\frac{1}{2}$ of the page, $\frac{1}{3}$ of the page . . . , the original illustration is made to occupy all, $\frac{1}{2}$, $\frac{1}{3}$ . . . of whatever rectangle is selected. On the limiting dimension, usually the width, the author marks with blue pencil the size he desires, *e.g.*, "Reduce to $4\frac{5}{8}$ inches."

Balance in Line Drawings and Graphs

Line drawings and graphs are made with India ink, usually on tracing cloth, Bristol board, or the equivalent, and sometimes on celluloid sheets

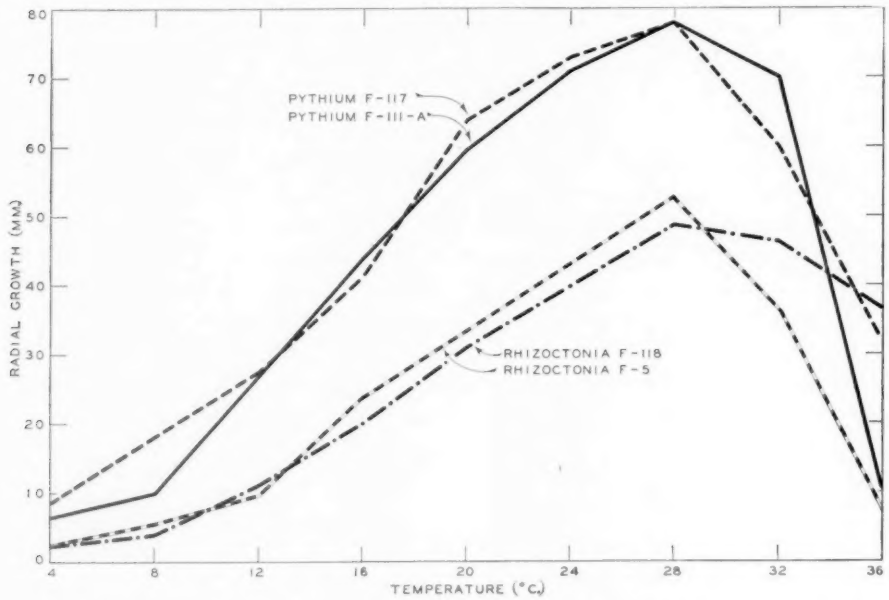

FIG. 2. Temperature and radial growth of four damping-off fungi on potato-dextrose agar in Petri dishes.

(24). The engravers prefer originals rather than photographic copies. The original is essential if a rectangle has been copied askew—and usually it has been.

The size and weight of letters, numbers, lines, and dots used in the origi-

[3] For 1946 a page of PHYTOPATHOLOGY measures $4\frac{5}{8}$ by $7\frac{1}{2}$ inches; of the JOURNAL OF AGRICULTURAL RESEARCH, $4\frac{7}{16}$ by $7\frac{3}{8}$ inches; and of the AMERICAN JOURNAL OF BOTANY, $6\frac{1}{4}$ by $8\frac{3}{4}$ inches, with 2 columns 3 inches wide.

nal drawings and graphs require planning, particularly when reduction is contemplated. A guide appears in figure 1.

Graphs and tables are used to present numerical data. Graphs are not only simpler and easier to comprehend than tables, but also they are cheaper to set. Since the style in graphs varies enormously, the beginner may read Anderson's (3) discussion and examine the best graphs in recent publications for acceptable methods.

An example of a relatively simple graph is in figure 2. These data are representative of 3 separate trials; the results of 2 are omitted. Although short, the legend makes the graph clear without reference to the text and with little, if any, repetition of the text. This graph was made with India ink on Bristol board. The size of the original was calculated by measuring the space it should occupy on the printed page and by making the original 4 time as large. The letters and numbers were made with commercial lettering guides but printed letters of the right size may be used. For best appearance the white area between letters needs to be approximately the same in all cases. This means that a C and O must be nearer together than an M and N. The letters and numbers were large enough on the original (for the actual size of the original, make comparisons on figure 1) to read even after reduction, which practically eliminated certain imperfections obvious on the original. Letters smaller than this on the printed page are too hard to read. The spacing of critical points of the scales on the side and bottom is proportioned according to a definite arithmetic (or geometric) progression. Equal spacing between numbers is not acceptable in a series like the following: 2, 4, 10, 16, 20, 50, 100, 150.

There are no vertical and cross lines in the graph proper in this case because a few millimeters or a degree or two are not important, and because the general situation is observed more easily this way. However, the beginnings of such lines do appear for clarity and for anyone who wishes to complete them. A few light guiding lines may be helpful sometimes where the exact reading is critical or if they do not distract from the essential features. The meaning of each graph line is indicated right at the line itself, which is heavier than the scale lines. Four graph lines are easily followed, but more than 5 or 6 become confusing. A continuous graph line between actual points of measurement is justified in this figure because measurements at intermediate points would probably fall near or on the line. However, in a different experiment, if a series of grain varieties or chemicals was placed at the bottom, and percentage of infection at the side, vertical bars instead of continuous lines would be needed because there are no intermediate points. This is discussed in more detail by Anderson (3). He also explains that the preparations of such bars and other conventionalized figures are frequently easier when suitable cross hatching on transparent film (secured from any good art store) is applied properly.

TABLES HAVE MANY POSSIBILITIES

Data too complex or discontinuous for graphs or for text discussion often

appear best in tables. Since their typesetting cost is 2 or 3 times that of ordinary composition, they should be used frugally and only when well organized.

While the acceptable styles vary enormously, an example (Table 1) may be useful. The data are representative, and more similar data have been omitted. The table has been prepared with maximum simplicity. The legend is short, but it is clearly worded, as are the column headings, so that the reader will understand the table without reference to the text. There is little, if any, repetition in the text of data given in the table. The columns have been arranged to fit on the printed page.[4] Only the headings, proper names, and items in the left column begin with capital letters. Units of quantity (days, number, per cent, etc.) may be set in italics at the top of figure columns. Negative results are indicated by ciphers, absence of data by dots. Only those decimal points appear which are clearly significant.

[4] The space occupied by letters and numbers can be estimated by the 8 point type in the following lines:

abcdefghijklmnopqrstuvwxyzabcdefghijklmnopqrstuvwxyzabcdefghijklmnopqrstuvwxyzab
ABCDEFGHIJKLMNOPQRSTUVWXYZABCDEFGHIJKLMNOPQRSTUVWXYZABC
12345678901234567890123456789012345678901234567890123456789012345678901234567890123456789

By counting letters, numbers, and at least 1 character for the space between columns, one can estimate the space a table will occupy. The large number of different possible arrangements, discussed in detail by Anderson (3), enables one to arrange almost any material to fit the page. For example, with 3 criteria of classification, A, B, and C, there are 12 possibilities, thus:

	A	A
C		
b		
b		
C		
b		
b		

	B	B
C		
a		
a		
C		
a		
a		

	A	A
	b	b b b
C		
C		

	B	B
	a	a a a
C		
C		

	C	C
A		
b		
b		
A		
b		
b		

	B	B
A		
c		
c		
A		
c		
c		

	C	C
	b	b b b
A		
A		

	B	B
	c	c c c
A		
A		

	A	A
B		
c		
c		
B		
c		
c		

	C	C
B		
a		
a		
B		
a		
a		

	A	A
	c	c c c
B		
B		

	C	C
	a	a a a
B		
B		

Non-significant decimals indicate a lack of discrimination. When they are used, they are uniform in a single column. "Ditto" is indicated not by ditto marks (") but by Do . . . or . . . do . . . , respectively, in the first or later column. Footnotes often supply information otherwise requiring a column. They are indicated by raised small letters. However, too many footnotes may be confusing. The authors can find in the literature many good examples to follow.

TABLE 1.—*Distribution in 1936 of Pythium and Rhizoctonia on red pine seedlings in Wisconsin forest nurseries*

Location	Soil		Seedlings plated	Platings positive[b]	Yield of positive platings	
	Type	Reaction[a]			*Pythium*	*Rhizoctonia*
		pH	*Number*	*Number*	*Per cent*	*Per cent*
Rhinelander	Vilas sand	5.0[c]	8	1	0	100
Port Edwards	Plainfield sand	5.0[c]	56	54	0	100
Do	do	5.1[c]	82	81	19	81
Gordon	do	5.2[c]	13	7	0	100
Madison	silt loam	5.3	17	15	20	80
Trout Lake	Vilas sand[d]	.....	19	10	80	20

[a] Determinations were made with a quinhydrone electrode and checked with a glass electrode.

[b] Positive plates yielded either *Pythium* or *Rhizoctonia;* all other plates were negative or yielded saprophytes or dubious parasites.

[c] Treated at seeding with 2 per cent sulphuric acid to control damping-off.

[d] Instead of soil from a nursery, this was soil from a nearby woods.

Particularly in a summary table when statistical analysis has been employed, one should mention the procedure and give an indication of the variability in the results; for example, the minimum significant difference or a summary of the analysis of variance.

Editors usually will take details of some representative data which are necessary to explain the procedure. After this they accept only summarized or well-digested data which give the results. Obviously, an author cannot publish all the data that have accumulated.

The tentative completion of tables, text figures, and, in addition, the pertinent literature citations before planning the details of the manuscript frequently saves considerable time in revision.

THE OUTLINE MARKS THE WAY

The preparation of a manuscript, just as with the itinerary for a trip, begins with a detailed plan, which frequently has been marked out already in the research program. The kinds of plans vary, but they should indicate the most suitable way, like road markers, and avoid meandering lanes. Most writers, unless they have something better, follow a conventional outline such as the following:

Title. This desirably is concise, descriptive, and favorable to accurate

indexing.[5] Thus another investigator is more likely to find it in his search of the literature. Further discussion is given by Oberly (14).

Introduction. This part often contains (1) a statement of the general subject, (2) the orientation, setting, and foundation on which the present investigation was made, but not a general literature review, and (3) the purpose of the studies described.

Materials and methods. This section usually explains broader aspects of what was used and how the work was done. However, certain details about individual experiments may fit in better in the specific section where they are described. The validity of research has its foundation on the method followed by the investigator. The validity of his technique and the logic of his interpretations need to be clearly apparent and must be acceptable (cf. Rawlins, 17; Riker and Riker, 19).

Observations, experiments, and results. This is the main part of the manuscript, containing brief descriptions of observations and experiments. Representative data and summaries of results should appear in a clear, concise, and logical form. Unimportant experiments and results, details of duplications, or data that cannot be interpreted are left out. Various subheadings may be useful. Under each the data may be presented in their logical order and without regard to the sequence in which the experiments were performed.

Discussion. In this portion the writer may answer the question "So what?" as he interprets the various data in relation to one another and to the foundation given in the "Introduction." He clarifies the meanings and implications of the various results and may indicate possible future developments. The reasoning done must be accurate and in accord with a recognized method of logic. Although in this place he can take a panoramic rather than a keyhole view of the situation, he wisely avoids wringing out of the results the last drop of theoretical implication. Summarization is avoided. If there is nothing important to discuss, this section should be omitted.

Summary. This is a brief résumé of the results together with the important conclusions, and not a general statement of the field of study. In a "note" occupying less than two printed pages, the last paragraph serves as a summary.

Literature cited. Five or more references are placed in this separate section. Four or less appear as footnotes.

Such an outline, and particularly "Observations, Experiments and Results," may be elaborated until every separate item of the paper has an entry. If the author does this in detail, he saves much time in later reor-

[5] For example, "A new oak disease" is inadequate. "The general importance, differential symptoms, and causal fungus responsible for a new wilt disease severe on red and black oaks" is too long. "The significance, symptoms, and cause of oak wilt" covers the essential points. "Studies on . . ." is omitted because it is implied before almost every title. The Latin name of the fungus is usually omitted, especially if its common name or the name of the disease appears. If the Latin name is used, the authority is omitted in the title, but is given in the text where the name first appears.

ganization, since he can hold in mind the divisions of the subject and their relations to one another more easily during the outline stage. The actual writing of the paper moves along better because final units in the outline furnish the subjects for the paragraphs. Some writers develop the outline in such a way as to provide not only the topics but also the topic sentences of the paragraphs. At first, this procedure may result in mechanical writing, but, in time, this is easily overcome. Even so, it is better than disorganization.

PREPARATION OF THE TEXT

Writing a manuscript need not be the disagreeable task often made of it. There is a technique for writing just as there is for making single-spore isolations—except that writing takes more time and patience to learn. However, once the skill is acquired, it can be a delightful means of self-expression.

A Writer Has Responsibility

An investigator stands before his colleagues and the public as an educated man and has the obligation to show it in his writing. He makes correct use of grammar, rhetoric, and logic; he keeps his readers in mind; and he shows them the consideration and courtesy of simple and clear exposition. He does not string together ponderous phrases without continuity, like wash on a back-alley line.

The aim of publication is to increase knowledge (*e.g.*, interpret the past, take care of the present, and provide for the future) and to leave the field clearer than it was. The last calls for the proper balance throughout of unity, coherence, emphasis, and accuracy. It is necessary to use language that not only gives the information but also gives it so clearly that it cannot be misunderstood. "Every paragraph and every sentence in your paper should receive careful and repeated consideration, first, as to whether it tells the exact truth; second, as to whether it is absolutely clear, *i.e.*, will convey the same meaning to all as to yourself (try it on your friends, if they will submit to it); third, as to whether it is complete, or requires various additions or qualifications—science is an eternal qualification; fourth, as to whether the sentences in it are entirely logical and move convincingly toward your final conclusions. These things can be determined only by repeated readings and much pondering" (21).

A publication is not judged by its length but (1) by the message it carries, (2) by the amount of well-directed and well-digested work it represents, (3) by the discrimination shown in distinguishing between the important and the relatively unimportant, and (4) by the conciseness in presenting the essential features. As a rule the more definitely a fact has been established by an investigator the more directly and simply it can be presented. The doubtful facts have to be hedged about with explanations, qualifications, and cautions. So brevity is an important quality.

A Premium on Brevity

Many writers use roundabout, wordy expressions[6] and include much unnecessary detail. Then they are puzzled when reviewers say, "Condense the manuscript to one-half its present volume." Such writers may think they are asked to omit many of their experiments and may feel resentment and frustration.

Among the diseases of manuscripts, a diarrhea of words is perhaps the most common. A representative case may clarify the symptoms and cure. Do you think the following paragraph, about the propagation of white pine selected for resistance to blister rust, is exaggerated? If so, you should see some examples collected from papers actually submitted.

"The multiplication of resistant white pine trees by means of making grafts has been already demonstrated as a feasible possibility. On an experimental basis, as was explained earlier, over 1000 grafts have been made and grown successfully during these studies in both green house and field conditions. Yet a grafted tree has been found relatively expensive because of the necessity, according to present technique, of using green house space in the winter time and cold frames in the spring. Counting scions, stock, pots, labor, *et cetera*, each graft is estimated to cost about 12 cents. This cost might be reduced somewhat if further research will be done to further improve the methods employed. It might also be reduced by puting this operation on the basis of a large scale commercial production. Even at this relatively high figure grafting may now be done as a practical possibility due to the high price paid for certain types of ornimental planting. But the most promising means that has appeared of reducing the cost of propagating rust resistant white pine trees is through the rooting of cuttings." More things than cuttle fish hide themselves in their own ink (cf. Maverick, 12; Barzun, 5).

This paragraph can be reduced to about one-third the space by omitting unimportant detail, can be clarified, and can be corrected (*e.g.*, it contains a dangling participle, some awkward phrases and four misspelled words) as follows (cf. Riker *et al.*, 20):

"Grafting white pine is feasible for experiments. Yet grafted trees are relatively expensive now (about 12 cents each) because they are propagated

[6] For example, some expressions with their shorter equivalents follow:
it will be seen from the foregoing figures = these figures show
it would thus appear that = seemingly or apparently
it is true that = admittedly
it is this that = this
the fact that the cultures grew shows = the growth shows
the question as to whether = whether
during the time that = while
at an earlier date = previously
with reference to = about or concerning
the treatment having been performed = after treatment
under greenhouse conditions = in the greenhouse
goes under the name of = is called
plants exhibited good growth = plants grew well
conducted inoculation experiments on = inoculated

in a greenhouse. Even so, grafting is possible for certain types of ornamental planting. Although improved technique and large-scale commercial production might reduce this cost, it will doubtless remain higher than that for cuttings."

Such condensation is prized by scientists who seek as many facts and ideas as possible in ten minutes of reading—but it may be anathema both to journalists writing at so much per word and to readers seeking effortless entertainment.

The rearrangement of words and phrases is really an interesting game. Of course, manuscripts can be cryptic and lose precision if condensed too much. While many adolescents might disagree, it would be a shame to reduce Caesar's "Commentaries" to Sherman's "War is hell." The beginner might try condensing manuscripts written by others[7] until he discovers how much fun it is and how to do it well. It is much more difficult for him to see and to correct the weakness in his own composition.

Being Dull or Interesting

In the choice and arrangement of words lies the difference between dull and interesting composition—providing one has something to say. Being dull involves, for example, (1) numerous clichés and platitudes, (2) verbosity and circumlocution (For goodness sake, come to the point!), (3) obscurity (What does he mean, anyway?), (4) the excessive use of the passive voice, (5) long pretentious words in stilted phrases,[8] and (6) expressions that do not fit together. All of these can be overcome with a little care. While perhaps no one would introduce into a report on potato late blight the lure, suspense, and drama of love and murder, still technical writing need not make us yawn. For example, one can at least aim at satisfying organization, directness, brevity, lucidity, concreteness, and even an occasional daring or sinewy phrase. Words and expressions that suggest pictures and actions help to hold interest and to clarify meaning. Overstreet (15) has developed further this important subject.[9]

Accuracy—a Prime Necessity

The definitions of words are frequent sources of trouble (cf. Committee, 8, 9). Words are the most valuable of scientific tools; they convey facts and ideas to others; but they can be dangerous. Occasionally some one extends a meaning without bothering to see whether it is valid, and thus he has a false sense of security. Many an argument has developed over different concepts for the same word. For example, "single-cell culture" applied to bacteria means for one person a culture from a single bacterial colony in a Petri dish (and thus no direct evidence that it is free from mixture), while

[7] If you do try it, please use discretion. It may be no safer to criticize a man's brain child than to slap his little Tommie or Susie.

[8] They remind Maverick (12) of the ridiculous strutting pomposity and the "gobbledygook" of his turkey gobbler.

[9] We have just seen a new and excellent aid to writing for easy reading. It is by Rudolph Flesch, "The art of plain talk," 210 pp., New York [1946].

for another it means a culture derived from an observed and a mechanically picked individual bacterium. If there is danger of a critical word being misinterpreted, the author may wisely explain just what he does mean.

Emphatic expressions about conclusions are undesirable because of the space they occupy and the suspicions they arouse. The clearly stated fact needs no reenforcing. If a writer says, "The data clearly show beyond possible question that . . . ," it suggests that he needs to convince himself. It reminds the reader of the timid boy who whistled to keep up his courage.

Teleological statements always arouse criticism. Two sentences may serve as examples: "The stomata closed in order to reduce further water loss from the leaves." "The germ tubes formed appressoria on the cuticle to help send infection pegs into the tissue." These expressions, which suggest that stomata and germ tubes had a purpose for what they did, indicate illogical thinking, careless composition, and especially departure from the approved scientific attitude.

Trade names of fungicides are far from precise. They have appeared on materials that have been changed again and again in composition. Even when the container gives the formula, another investigator, especially in future years, may not know exactly what it was. Such trade names should not be used unless the chemistry of the product is given. When used they are capitalized (Semesan, Vigoro, Hudson sprayer . . .).

Accuracy is a critical requirement in technical writing. The scientist must scatter the fog of abstract or obscure expressions and let the light shine clearly on the concrete base of reality. If he is careless in reporting his work, he may be suspected of being careless in his experiments, in taking data, and even in thinking about them. The design and interpretation of the experiments need to be clearly in accord with logical scientific methods (cf. Wolf, 27). Lucidity is the sovereign politeness of the writer; he must make sure that his words can be given no unintended other meaning.

To read accurately seems to be among the hardest things in this world. To misunderstand is among the easiest—because we tend to get the idea we expect to find, or we wish to find; and too often we may twist the meaning to make it what we like (cf. Barzun, 5). Poor writing makes hard reading. Repeated lack of precision through laziness or scatterbrains suggests the adulteration of food; there is possible danger and certainly bad taste.

A clear and sharp line needs to be drawn between fact and opinion, between reason and wishful thinking. The scientist should be objective and should formulate only conclusions justified by the evidence. Deductions from preconceived notions or, much worse, from prejudice, indicate a mind closed to impartial and honest reasoning. Sooner or later they detract from a man's standing and from scientific progress. Administrative pressure or personal ambition may lead to the misinterpretation or over-enthusiastic reporting of a valid new discovery. Examples are common. The salvation of the plant world has been announced repeatedly by extravagant and unjustified claims for such valuable items as fertilizers, sprays, seed treatments,

hormones, and a long list of others. When an investigator extends a generalization, he is probably also extending his neck. He may well recall that "no generalization is true—not even this one."

One needs to be objective and to show consideration for his colleagues, especially when they are in error. Comments about one's own research and that of others are in bad taste when personal, and in good form when factual. Disparaging statements commonly boomerang to the disadvantage of the writer. While spectators may enjoy the free show, who can throw mud and still keep his hands clean? Although Erwin F. Smith practically macerated Fisher in their well-known controversy, Smith lived to be ashamed of the way he did it. Difference of opinion and good-natured controversy are healthful and stimulating, but "dog fights" are bad. Most conflicts can be settled best through correspondence, in conference, or by further experiments without wasting space in print. After all, the question is not, "Who is right?" but "What is true?"

This objective approach, when applied to earlier contributions by others, allots to each the treatment it deserves in relation both to the present contribution and to the subject as a whole. A nationalistic or a personal twist of the credit assigned to earlier work, either by omission or by minimizing its significance, can only corrode the writer's reputation. The white radiance of established fact shines more beautifully if it is unsullied either by the sweat and blood of acrimonious controversy or by the odor of piffling jealousy and unenlightened ambition.

Bad and Good Beginnings

Novices in technical writing frequently get poor results for several reasons: (1) They have trouble writing a good "Introduction." This is one of the most difficult parts of a paper. Even when the statement of the problem, the scope of the work, the background, and the purposes are clear from the outline, beginners often have trouble. If they do, they may well pass over it lightly or even skip to "Materials and Methods,"—perhaps the easiest part for the initial effort. (2) They have not finished, at least in preliminary form, the text figures or tables and have insufficiently digested their results. They begin to write before they have decided which results should be left out, which used, and what they mean. (3) They may be discouraged because in the first draft they seek perfection; therefore, they may wreck the train of thought on the spelling of "desiccate," a dangling participle, or the choice between 50 cc. and 50 ml. (4) They allow insufficient time for writing. When there is a deadline date, the pressure increases the difficulty. For example, after reviewing the literature and after completing and digesting the experiments, a graduate student should allow 3 entire months for writing his doctor's thesis. Even experienced and skillful writers seldom produce an excellent manuscript without going over it many times, without having several clean copies made, and without laying it aside to cool before final revision.

A happy way to begin is to recognize that for the first draft the all important thing is to transfer the thoughts to paper. With an outline to serve as a guide down the main line and away from sidetracks, one can run lightheartedly and with disregard for spelling, punctuation, and rhetoric which might wreck the train of thought. At this stage the self-expression of personality appears which provides style and raises an otherwise dull subject to the level of pleasant reading. So long as the "spirit moves," no question of detail is permitted to interfere. Some people find it helpful to imagine several listeners and to write as if talking to them. They may include everything possibly significant and later eliminate nonessentials during revisions of separate parts. It is easier to cross off than to make inserts.

Paragraphs Are Single Units

Many writers place a single paragraph on one sheet of paper to avoid the clutter of cutting and pasting during later revisions. Then they can subtract, add, or move a single unit without disturbing other sections.

The composition of a paragraph varies with different circumstances, but desirably contains only one central idea. An investigator may consider a structure something like this: (1) topic, in which the subject, but not the conclusion, is stated; (2) explanation of topic, in which appear any necessary clarifications of terms or greater descriptions of various items; (3) central part, in which is given the detail regarding the topic discussed; (4) conclusions, in which the writer's estimate of the meaning of this detail is explained; and (5) transition, in which the connection between this paragraph and the one following is shown. The transition may appear as a word or phrase in the first sentence of the next paragraph.

The chief objection to this form is a tendency to be stilted unless the transitions are skillfully handled, but this is a much smaller fault than obscurity. The topic sentences sometimes cause difficulty because, instead of the topic, writers place the conclusion first; then they endeavor to justify it by the material that follows. This suggests an effort to prove a preconceived idea rather than to make an impartial evaluation of the evidence. Herein lies one of the basic differences between technical and news writing: a reporter tells the important thing first; he gives various details later.

The chief advantage of writing so that the topic is clearly stated in the first sentence (and better still in the first phrase of the first sentence) is that the reader can glance rapidly over the article; he can locate easily the paragraphs in which he is interested: he can skip everything else. And this is, after all, a critical test for good writing in a technical paper. This system is a little hard at first. But once a writer develops the habit, he saves much time in hunting through the manuscript for a particular item, especially after a clean copy has been made. Likewise, readers can glance rapidly through a stack of papers written in this way. Articles not so prepared they may file away for reading in a leisurely time—which never comes.

Admittedly, there are other approaches. However, technical writing

has certain limitations. They prevent the writer from employing some of the interesting techniques useful in newspapers and popular magazines. For example, (1) a scientist must use the technical jargon (the shorthand) of his subject. It is required for brevity, clarity, and precision, even though it may be unintelligible to those untrained in this subject. (2) He needs to be brief. (3) He should avoid even the appearance of personal bias and should state first the facts and then draw the conclusions. (4) When he must walk in those fields where emotions blossom to influence judgment, he considers only the valid data and logical inferences from them. He shuns the assessment of values he cannot measure. He differentiates fact from opinion. Such conditions are rather incompatible with the "human interest" sought by journalists. So within rather exacting requirements each scientist will use the best way for his purpose. But he should not hope to find a "short cut" for painstaking revisions.

Revision Requires Time and Care

Once the first draft has been made, its revision and correction call for a different approach. A refresher glance at one of the many good handbooks of composition (*e.g.*, Woolley, Scott, and Berdahl, 28) is sure to be helpful.

The correction of the text involves many details which are frequently merely conventions adopted for convenience, uniformity, and precision. Terminology; spelling including capitalization, abbreviation, and compounding words; punctuation; and citations are a few of the questions requiring special attention. Except where otherwise stated, PHYTOPATHOLOGY aims to follow the Public Printer (16).[10] Attention is directed particularly to such abbreviations as: cc. for cubic centimeter (space); ml. for milliliter (volume, instead of cc.); gm. for gram; and μg. for microgram (instead of gamma)—all with periods. For spelling and common meanings of words, PHYTOPATHOLOGY follows Webster's New International Dictionary; for special definitions,[11] recent reports (8, 9); for common names of plants, Standardized Plant Names (2); and for scientific names, the International Rules of Botanical Nomenclature (7). However, dissenters have their rights, and most editors defer to authors with valid arguments. The description of a new fungus species should have a summary in Latin, and that of a new bacterial species, in English. Among others, Trelease and Yule (22) discuss the preparation of scientific and technical papers, and Woglum (26) has assembled a list of improperly used words.

An alteration in one place frequently calls for a survey of the text for

[10] For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C.

[11] Some technical words are hard to define. The committee reports commonly give the best definitions, but even they are more or less tentative. For example, among the various definitions for "plant diseases" we have this facetious one, "Plant diseases are the things plant pathologists work on." If you know what we mean, a definition is not necessary; if you do not, it does not help much. Are we reduced to saying, "Plant disease—you know what I mean"?

some distance, before and after the change, for corrections dependent on the first. This deserves special emphasis.

Should such details be left for the Editorial Board? Not until the Society pays its editors. Meantime, they perform an enormous and thankless "labor of love," and they deserve to receive manuscripts in good form.

The criticism of a manuscript by well-informed colleagues is eminently desirable. Even though it has been prepared with extreme care, has been reread critically by the writer a dozen or more times, and has received a number of retypings, the suggestions of colleagues are sure to reveal various rough places and to discover statements which might be construed in two ways. It is a common experience that a man cannot edit his own manuscript. The examination by others will guard against a misinterpretation of the data presented, against drawing conclusions from inadequate data, and against publishing a paper that may later be embarrassing, not only to the author but also to others in the same institution. Tabular and statistical matter particularly are to be checked. The values of such a procedure so far outweigh the extra labor involved that such a review is required by most editors.

Those Troublesome References

References to other publications present many opportunities for error. Just as editors frown on long literature reviews, so they discourage an excessive number of citations. Unless they are really necessary, a large number makes a paper appear pedantic rather than learned. How often we see a general statement followed by a string of citations when one or two would serve as examples.

An easy method, while preparing a manuscript, is to enter in the text the name of the author with the year of publication. If the same author has several publications in a year, they are designated by letters (*e.g.*, Jones, 1935a, 1935b, 1935c, . . .).

The complete citation may be placed on a card of convenient size, the reference cards being kept in alphabetical order. The form of the citation is a convention and follows the best form in recent issues. Examples appear at the end of this paper. In case of doubt, the plan suggested by Whitlock (25) is excellent. The abbreviations for the titles of the periodicals may be prepared and interpreted according to Bartholow's (4) list. Before the literature cited is typed double spaced from the alphabetically arranged cards, every letter and number of the citation should be checked against the original. Do you think this is a tedious, time-consuming job? Obviously. Should it be left to the editors? Certainly not.

Before the final copy is typed, frequently the author's name and always the year of publication are replaced by numbers,[12] corresponding to those in the alphabetically arranged "Literature Cited," providing there are 5 or more. Fewer citations are handled as footnotes. Once the list is done, any

[12] There are important arguments in favor of leaving the names of the investigators and the year of publication in the text (cf. American Journal of Botany).

deletion or addition of a reference means changing many numbers both in the text, where they are easily missed, and in the "Literature Cited." For example, if a reference to a paper by Brown is added or removed, all the numbers applying to papers by authors following Brown in the alphabet must also be changed.

THE FINAL COPY

The final copy is typed double spaced with ample margins on good white bond paper (preferably about 8½ by 11 inches) and with at least 2 carbon copies. Footnotes and legends for text figures and plates are typed immediately below the first notation to them and are set off from the text by a line above and another below. The printer likes a separate page carrying the legends of the text figures. Each table is typed on a separate sheet (so it can be taken out and given to a different typesetter) and inserted as a numbered page in the manuscript immediately after the page on which the table is first mentioned. The manuscript is accompanied by a table of contents showing the rank of the various center headings and a statement of the number of pages, tables, and text figures.

The carbon copies smear less and are more easily read if each page is heated momentarily above the melting point of the wax in the carbon paper. As this wax melts, it is absorbed by the paper. The wax impression smears easily while hot, but is fixed after cooling.

When this final typing and its carbon copies have been made and checked, the author still has several details to consider.

Before a manuscript is sent off for publication, it is hoped that the writer can answer "Yes" to the following questions: (1) Has every reasonable effort been made to find a flaw in the technique used and to discover any errors either in the observations and experiments themselves or in the conclusions drawn from them? (2) Is it reasonably certain that, when able investigators repeat the work, they must secure the same results and draw the same conclusions? (3) Has the size of the manuscript been reduced to the minimum compatible with clarity of presentation? (4) Have at least two qualified colleagues read and criticized the manuscript? (5) Has a competent statistician examined the tables and computations? (6) Do the illustrative materials, tables, and legends meet the requirements of PHYTOPATHOLOGY? Will each figure and table fit well on the printed page? (7) Is there a reference in the text to every table, to every figure, and also to every publication referred to in "Literature Cited?" (8) Has every entry in "Literature Cited" been checked against the original, and is the form of reference suitable for PHYTOPATHOLOGY? (9) Have you prepared a table of contents showing the relative rank of the different headings? Have you written on the first page the total number of pages, text figures, and tables? (10) When the paper was finished, was it laid aside for at least a month and then reexamined critically with a fresh viewpoint and without bias? (11) Has it been approved by the proper administrative office (if

this is required)? (12) Is PHYTOPATHOLOGY the best journal for this manuscript?

INCIDENTAL MATTERS

After a manuscript has been received by the editor, two anonymous but well-qualified referees examine it. The time is shortened when the author sends in a complete duplicate of his paper, except for complex illustrations. While reviewers do their best and are almost always helpful, they are human. The author must make sure that they have not introduced any mistakes. This editorial procedure seems necessary not only to maintain a satisfactory standard but also to assist the authors, so far as is humanly possible, to avoid serious errors.

For any important corrections suggested, the manuscript goes back to the writer. However, when it has only minor editorial changes, the manuscript goes to the printer and the author checks the changes when he reads the galley proof.

While no one needs to achieve perfection, he should meet at least a satisfactory standard. If he fails in this, his paper either will be delayed until he does accomplish it or will be rejected.

After galley proofs are received, errors are indicated by standard proof-reading marks (cf. Riker and Riker, 19; or Public Printer, 16). At this stage only essential alterations are permissible. Any minor changes in wording or punctuation are avoided. These, of course, should have been made earlier on the manuscript, when a few strokes with a pen would have accomplished the results.

Typesetter's errors should be marked in red ink only on the galley proof sheets. The original manuscript should never be altered when read against the galley proof. Author's alterations from copy should be indicated in black ink only on the proof sheets. The printer charges for the latter at the prevailing rate. The change of a single long word may require that the rest of the paragraph be reset.

Abstracts of papers for transmission to Biological Abstracts are requested at the time proof is returned to expedite their publication. Ordinarily, the abstract does not exceed in length 3 per cent of the original paper. A guide for preparing such abstracts is available (6).

Reprints are ordered at the time proof is returned.

SUMMARIZED RECOMMENDATIONS

As you prepare manuscripts for PHYTOPATHOLOGY, you are requested, as far as it is feasible, to follow these recommendations. If at first some of them resemble armchair fiats, please consider the explanation for most of them in the preceding pages.

Illustrations

1. Plan in relation to page dimensions and allow for the space needed for a legend.

2. Avoid duplication of illustrative material and an excessive amount. The author may be charged for illustrations and tables which combined occupy more than 20 per cent of the page space.
3. When there are several similar illustrations, combine them into one figure whenever possible, but do not combine photographs and line drawings.
4. Letter units of a composite illustration (A, B, C, . . .) on the picture itself. Letter specific parts of a unit (a, b, c, . . .). The author should employ either a skillful hand, a lettering guide, or suitable type. Do not leave this to the editor.
5. Submit only photographs that are glossy, have plenty of contrast, and have been mounted without wrinkles with rubber cement.
6. Trim away unnecessary parts of photographs (flower pots, backgrounds, operators, nonessential plant parts . . .).
7. Use undiluted India ink drawings or silvertones rather than poor photographs.
8. Prepare drawings for zinc etchings on Bristol board or the equivalent.
9. For graphs avoid green-line graph paper and typewritten labels or symbols. Use undiluted India ink on special blue-line paper, white paperboard, or tracing cloth.
10. Make sure of a heavy, even, steady line; of size, clearness, and openness of lettering or stippling; and of accuracy of scales—all in relation to the reduction planned.
11. If magnifications are indicated, calculate them for reduction of illustration when necessary.
12. Indicate with blue pencil the reduction desired.
13. Prepare legends that are brief but clear with little, if any, repetition of the text. Place author's name, title of manuscript, figure number, and legend on the back of the figure.
14. Pack illustrations well for mailing. Use plenty of corrugated paperboard or the equivalent to avoid wrinkles, folds, cracks, and dents from paper clips.

Tables

1. Avoid long, complex, or undigested tables.
2. Avoid too many tables—the most expensive part of a manuscript to print. The author may be charged for more than 20 per cent all told of illustrative and tabular material.
3. Use the text rather than small tables whenever possible.
4. Arrange the table to fit upright on the page ($4\frac{5}{8}$ by $7\frac{1}{2}$ inches in 1946) whenever possible.
5. Number tables with arabic numerals (1, 2, 3, . . .).
6. Provide a complete table heading for every table.
7. Provide clear and concise column headings.
8. Letter footnotes a, b, c, ...
9. Explain any symbols used in a table.

Text

1. Allow plenty of time for writing. Use a simple, direct style which is condensed, but not so condensed as to be cryptic or to sacrifice precision and clarity.
2. Organize the material following logical sequences and not according to the order in which experiments were performed.
3. Arrange the paper so that a reader can glance over it rapidly and can locate easily any special part he wishes.
4. Revise the manuscript until it has unity, coherence, emphasis, and accuracy, and until it is so clear it cannot be misunderstood.
5. Avoid pompous, pedantic, or wordy expressions and unnecessary detail. However, give all the facts necessary for a well-trained person to repeat the experiments.
6. Be objective regarding your own results and those of others. Avoid teleological statements.
7. Make sure that the reasoning follows a well-recognized system of logic. Likewise, be sure that in each experiment or test you used an acceptable method.
8. Avoid long literature reviews and an excessive number of citations, but do not ignore relevant work of others.
9. Distinguish clearly between statements supported by experimental evidence and those based on speculations.
10. Use headings and sub-heads, as well as good paragraphs, to enable the reader to "skim" the article for its general subject matter and to locate quickly any detailed part he seeks.
11. Provide a summary, usually not longer than 3 per cent of the paper, in which the contributions are stated clearly and succinctly in specific terms rather than in general statements of the field covered.
12. Let tabular data and illustrations speak for themselves. Confine the text discussion to the meaning of the data.
13. Avoid presenting data in duplicate or triplicate, *e.g.*, in graphs and tables, or in graphs, tables, and the text. Choose the best of the three forms and omit the others.
14. Although experiments must be done over and over to make sure of the results, present only representative or summarized data.
15. Explain new terms and terms not in general use. State the chemical composition of fungicides.
16. Provide a Latin summary with the description of a new fungus species.
17. Number tables, text figures, and footnotes (beginning each series with 1, 2, 3, . . .) consecutively throughout the paper.
18. Employ the conventions regarding, for example, abbreviations, plant names, and references to figures, tables, and literature citations.
19. Type the final copy with heavy black ribbon, with double space, and with ample margins on white bond paper. Approximately 8½ by 11 inches is the preferred size for paper. If the first carbon is sent with

the original, more rapid review may be achieved. Keep a carbon both for reference and insurance against loss.

20. Plan papers that will occupy less than 2 printed pages as "Phytopathological Notes."

Literature Citations

1. Handle 4 or less literature citations as footnotes.
2. Arrange 5 or more literature citations alphabetically according to author at the end of the paper under the heading "Literature Cited."
3. Provide a complete citation—author or authors, complete title, complete reference to journal, bulletin, circular, book. . . . For a journal give volume, limiting pages, and year of publication. For a book give edition, total number of pages, year of publication, and city where published.
4. In the body of the paper refer to literature citations by number. See that the numbers in the text and in the "Literature Cited" correspond without any left over.
5. Check every letter and every number in the literature citations against the original.

Finishing Items

1. It is hoped the author can answer "Yes" to the following questions:
   (a) Have you critically reexamined the methods, experiments, and conclusions?
   (b) Will further repetitions of the work yield the same results and conclusions?
   (c) Have you eliminated all unnecessary materials from text, tables, and figures?
   (d) Have two qualified colleagues criticized the manuscript?
   (e) Has a competent statistician examined the tables and calculations?
   (f) Have you met the conventional requirements regarding illustrations, tables, legends, and references to them? Will the text figures and tables fit properly on the printed page?
   (g) Is there a reference in the text to every table, figure, and item in the "Literature Cited"?
   (h) Have you checked every entry in the "Literature Cited" against the original?
   (i) Have you prepared a table of contents showing the ranks of various headings and sub-headings?
   (j) After the paper was finished, did you lay it aside for a month and reexamine it critically with a fresh viewpoint and without bias?
   (k) If required, has the proper administrative officer approved it?
   (l) Is PHYTOPATHOLOGY the best avenue for publication?
2. After two anonymous reviewers have criticized the manuscript, it will be returned to the author, if necessary, for his further consideration.

3. The author may be charged for an excessive number of tables, line cuts, and half tones if such materials all together exceed one-fifth of the total number of pages.
4. Authors will make corrections promptly on the galley proof (not on the original manuscript). Mark typesetter's errors in red ink and author's changes from copy in black ink. Return the proof promptly and send back with it two copies of an abstract and any order for reprints.
5. The author will be charged for any excessive changes of proof from the text.
6. Care in observing these conventions and suggestions will help the editorial staff and will enable colleagues to share the pages available in the journal.

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LITERATURE CITED

1. ALLEN, E. W. The publication of research. 19 pp. [Washington, D. C. Mimeographed reprint.] 1945.
2. AMERICAN JOINT COMMITTEE ON HORTICULTURAL NOMENCLATURE. Standardized plant names. Ed. 2, 675 pp. Harrisburg, Penn. 1942.
3. ANDERSON, J. A. The preparation of illustrations and tables. Trans. Amer. Assoc. Cereal Chemists **3**: 74–104. 1945.
4. BARTHOLOW, FRANCES A. Abbreviations employed in Experiment Station Record for titles of periodicals. U. S. Dept. Agr. Dept. Bull. 1330. 1925.
5. BARZUN, J. Teacher in America. 321 pp. Boston. 1945.
6. BIOLOGICAL ABSTRACTS. Guide for collaborators in the preparation of abstracts for Biological Abstracts. Ed. 2, rev., 12 pp. Philadelphia, Penn. 1928.
7. BRIQUET, J. International rules of botanical nomenclature. . . . Compiled by the Editorial Committee for Nomenclature from the Report of the Subsection of Nomenclature. Ed. 3, 151 pp. Jena. 1935.
8. COMMITTEE ON STANDARDIZATION OF FUNGICIDAL TESTS. Definitions of fungicide terms. Phytopath. **33**: 624–626. 1943.
9. COMMITTEE ON TECHNICAL WORDS. Report of the Committee on Technical Words. Phytopath. **30**: 361–368. 1940.
10. EVANS, M. W. Making photographs of plants to be used as illustrations for scientific papers. Jour. Amer. Soc. Agron. **17**: 526–532. 1925.
11. GROVES, A. B. A photographic light box for use in agricultural research. Jour. Agr. Res. [U.S.] **44**: 467–475. 1932.
12. MAVERICK, M. The case against "gobbledygook." N. Y. Times, Mag. Sect. May 21, 1944, pp. 11, 36, and 37. 1944.
13. NAYLOR, E. Pen and ink drawings from photographs. Science **73**: 15. 1931.
14. OBERLY, E. R. Abstracts and titles of scientific articles from the librarian's standpoint. Science **54**: 491–492. 1921.
15. OVERSTREET, H. A. The psychology of effective writing. *In* Influencing human behavior. pp. 87–109. New York. 1925.
16. PUBLIC PRINTER. United States Government Printing Office style manual. Rev. ed., 435 pp. Washington, D. C. 1945.
17. RAWLINS, T. E. Phytopathological and botanical research methods. 156 pp. New York and London. 1933.
18. REDDICK, D. Report of the chief editor. Phytopath. **8**: 182–190. 1918.
19. RIKER, A. J., and REGINA S. RIKER. Introduction to research on plant diseases. 117 pp. Madison, Wis. 1936.
20. ———, T. F. KOUBA, W. H. BRENER, and L. E. BYAM. White pine selections tested for resistance to blister rust. Jour. Forestry **41**: 753–760. 1943.
21. SMITH, E. F. An introduction to bacterial diseases of plants. 688 pp. Philadelphia and London. 1920.
22. TRELEASE, S. F., and E. S. YULE. Preparation of scientific and technical papers. Ed. 3, 125 pp. Baltimore, Md. 1936.

23. WEISS, P. Biological research strategy and publication policy. Science **101**: 101–104. 1945.
24. WESTON, W. H., JR. The use of celluloid in botanical illustrations. Amer. Jour. Bot. **22**: 384–391. 1935.
25. WHITLOCK, CAROLYN. Citations to literature in the Journal of Agricultural Research, technical bulletins, circulars, and miscellaneous publications (other than bibliographies). U. S. Dept. Agr. [1940.]
26. WOGLUM, W. H. Please! An editorial. Cancer Res. **2**: 846–850. 1942.
27. WOLF, A. Essentials of scientific method. Ed. 2, rev. and enl., 174 pp. London. 1928.
28. WOOLLEY, E. C., F. W. SCOTT, and E. T. BERDAHL. College handbook of composition. Ed. 4, 444 pp. Boston. [1943.]

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PHYTOPATHOLOGICAL NOTES

A Pycnidial Strain of Macrophomina phaseoli.[1]—During the course of work on root rots of snap beans in Georgia, a strain of *Macrophomina phaseoli* (Maubl.) Ashby which produces pycnidia and pycnospores abundantly on agar media was isolated. This pycnidial strain (culture No. 75) was obtained from infected hypocotyl tissue of a snap bean seedling having symptoms of charcoal rot at Experiment, Georgia, on May 25, 1944. The isolate was typical of *M. phaseoli*, forming a black crust of sclerotia over the surface of the medium. In about two weeks pycnidia appeared among the sclerotia. Pycnospores embedded in a viscous fluid oozed from the pycnidia and collected in white, opaque droplets plainly visible to the naked eye. Fifty pycnospores taken from a culture on three per cent malt agar measured 14.0–30.8 × 7.0–11.2 μ. A year later 20 single pycnospore isolations were made from this strain. All of these isolates were similar to the original strain; and all produced pycnospores on agar media, indicating that the strain was genetically stable.

Among the 58 isolates of *Macrophomina phaseoli* which have been examined in this study, culture No. 75 is the only one that has produced pycnidia on agar media. Attempts were made, therefore, to find some means of inducing the other isolates to form pycnidia in culture. The most satisfactory method tried was the following: Dried snap bean stems were cut in 10-inch lengths and soaked thoroughly in water. The stems were then inserted in large culture tubes 12 inches long, their basal portions being immersed in water to a depth of two inches. After sterilization, the bean stems were inoculated with various isolates of the fungus. The majority of the isolates formed scattered pycnidia somewhere along the bean stems within one month. The pycnidia were often elongated, compound, coralloid structures projecting above the crust of sclerotia. By this method, 35 different isolates of *M. phaseoli* were tested for the production of pycnidia. Of these isolates, 17 were from Georgia and 18 were from various other states. Three of the isolates from Georgia were furnished by Dr. J. L. Weimer, one from Georgia by Dr. G. E. Thompson, four from California by Dr. W. C. Snyder, one from Maryland by Dr. R. W. Leukel, two from Nebraska by Dr. J. E. Livingston, and one from California, four from Texas, and six from Oklahoma by Dr. E. C. Tullis. As is shown in table 1, only 20 of these isolates formed pycnidia. The pycnospore dimensions recorded in the table were obtained from measurements of 25 pycnospores from each culture. The isolates from sorghum appeared to have been maintained in culture for some time and to have degenerated to forms that produced an abundance of light-colored aerial mycelium and few sclerotia. Changes in the fungus produced by growth on artificial media may explain the failure of these isolates to produce pycnidia. Most isolates of *M. phaseoli* undergo such changes in

[1] Paper No. 151, Journal Series, Georgia Experiment Station.

culture. It seems desirable, therefore, to work with freshly isolated cultures whenever it is possible. It must be noted, however, that of the 17 strains from Georgia, the majority of which were freshly isolated, three failed to form pycnidia in culture under any conditions. Nevertheless, it was possible by this method to demonstrate the ability of most of these isolates of *M. phaseoli* to form pycnidia, although only a single isolate of the fungus formed pycnidia on agar media.

TABLE 1.—*Dimensions of pycnospores produced in culture on snap bean stems by various isolates*[a] *of Macrophomina phaseoli*

Culture No.	Place of origin	Host	Dimensions of pycnospores
			Microns
20	Georgia	Cowpea	.....
21	do	do	14.0–28.0 × 6.2– 8.4
24	do	Lima bean	14.0–28.0 × 5.9– 8.4
25	do	*Cassia nictitans*	19.6–28.0 × 8.1–11.8
43	do	do	.....
27	do	*Crotalaria intermedia*	19.6–26.6 × 8.4–10.6
28	do	*Lespedeza sp.*	16.8–22.4 × 6.2– 9.0
29	do	*Lactuca scariola*	16.8–33.6 × 7.8–11.2
45	do	Coffee weed	14.0–33.6 × 8.2–11.2
46	do	do	16.8–26.6 × 8.4–11.2
70	do	Sweet potato	.....
34	do	Snap bean	21.0–29.4 × 7.0– 8.4
35	do	do	16.8–33.6 × 6.2– 9.0
36	do	do	16.8–30.8 × 8.1– 9.8
37	do	do	14.0–22.4 × 7.0– 8.4
38	do	do	16.8–26.6 × 6.2– 9.8
75	do	do	11.7–22.4 × 8.4–11.2
31	California	Cantaloupe	16.8–33.6 × 7.8–11.2
32	do	Red Kidney bean	19.6–30.8 × 6.2–11.2
33	do	Bean	.....
42	do	Tomato	19.6–30.8 × 6.2–11.2
85	do	Sorghum	.....
30	Maryland	Sun flower	.....
41	Nebraska	Corn	19.6–30.8 × 7.8–11.2
88	do	Sorghum	.....
39	Texas	Cowpea	16.8–28.0 × 8.1–11.2
40	do	Sorghum	14.0–21.0 × 7.8– 9.8
84	do	do	.....
86	do	do	.....
87	Oklahoma	do	.....
89	do	do	.....
90	do	do	.....
91	do	do	.....
92	do	do	.....
93	do	do	.....

[a] Fifteen isolates failed to produce pycnidia in culture.

Inoculations with pycnospores from culture No. 75 were made on Tendergreen snap bean plants of all ages from the seedling stage to full maturity. Drops of a suspension of pycnospores were placed on nonwounded stems and leaves. After allowing the inoculum to dry, the plants were placed in a moist chamber for 48 hours. They were then returned to benches in the greenhouse. Lesions appeared on the stems and leaves within two weeks,

resulting in the death of leaves and portions of branches and, ultimately, of entire plants. Excellent results were consistently obtained from inoculations with pycnospore suspensions when other methods of inoculation failed. Inoculations through the soil with oat cultures of *Macrophomina phaseoli* were successful only when the soil was inoculated prior to planting. The seedlings then became infected during emergence. It seems probable that more satisfactory results from inoculations with *M. phaseoli* on other crop plants might be obtained, if inoculations after emergence were made with pycnospore suspensions.

Since use of this pycnidial strain has greatly facilitated inoculation experiments with *Macrophomina phaseoli* on snap bean, it is offered to pathologists who are working on diseases of other plants caused by this organism in the belief that it may be helpful to them. Cultures may be obtained upon request to the Georgia Experiment Station.—E. S. LUTTRELL, Georgia Experiment Station, Experiment, Georgia.

Resistance to Cercospora apii Fres. in Celery (Apium graveolens var. dulce).—The early blight disease of celery caused by *Cercospora apii* Fres. is one of the most destructive and widespread diseases of this crop. It occurs throughout the United States, but is particularly destructive in the southern states. In Florida it is necessary to apply Bordeaux mixture or other protective fungicides 12 to 16 times during the growth of the crop to control the disease.

Variety trials over a period of years at the Everglades Experiment Station, Belle Glade, Florida, have failed to disclose a commercial variety of celery sufficiently resistant that it could be grown without the use of fungicides. However, in 1940–41 forty-eight foreign collections of celery were planted at Belle Glade, Florida, and these were examined for resistance to early blight. The collection of varieties had been made by plant explorers[1] of the United States Department of Agriculture and had been released to the Everglades Experiment Station by the Department of Agriculture. Among this collection of foreign celeries there were seven varieties originally from Turkey which remained free of early blight lesions and produced vigorous plants. This was in contrast to the extreme susceptibility of all commercial varieties from the United States.

The resistant Turkish celeries were dark green, hollow-petioled, strongly flavored plants, closely resembling celeriac. They probably were types used for cooking rather than as a green salad. There was no possibility of developing promising new varieties by selfing of single-plant selections from these varieties.

Since climatic factors operate against successful breeding of celery in Florida, a number of plants from the seven early-blight-resistant varieties were taken up and shipped to the Department of Plant Breeding at Cornell University in March and April, 1941. Only one of these plants flowered

[1] F. L. Wellman and H. L. Westover.

during the summer of 1941 and an attempt to hybridize it with Cornell 19 failed. The other plants produced no inflorescence during the summers of 1941 or 1942. After storage in a cold-frame during the winter of 1942–43, the plants were taken into the greenhouse late in January. Two plants flowered in May, 1943, and were caged with Cornell 19 plants which were in flower. Flies were used to effect cross-pollination. Seed was obtained during the summer from each of the caged plants, and it was later learned that the following four crosses were established: PEI 115557 × Cornell 19, Cornell 19 × PEI 115557, PEI 120875 × Cornell 19, and Cornell 19 × PEI 120875.

The F_1 plants were grown in a greenhouse at Cornell University during the winter of 1943–44 and flowered in April, 1944. Nine F_2 lines from these crosses were grown in the field at Belle Glade, Florida, during the winter of

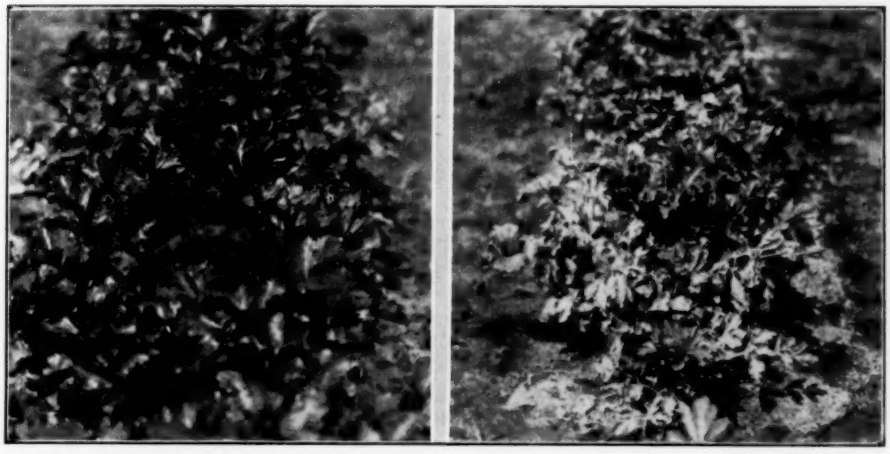

FIG. 1. An F_3 line of celery having a high degree of resistance to *Cercospora apii* (at left) and a susceptible commercial variety (at right).

1944–45. Most of the plants in these lines were very large, dark-green, hollow-petioled, and considerably more resistant to *Cercospora apii* than commercial varieties. There were a few plants which had segregated to solid petioles. These offered the opportunity long desired of combining disease resistance with a horticulturally acceptable plant type. Twenty-eight F_2 plants were selected as highly resistant to *Cercospora apii*. All of these had solid, fleshy petioles. Some were as green as Pascal celery, while others were a lighter green. Two selections were of the self-blanching type. The selected plants were shipped to Ithaca, N. Y., in March, 1945, and seed was obtained from seventeen of them during the summer. Nineteen other F_2 lines selected at Ithaca the previous summer also produced seed during 1945.

A planting of 35 F_3 lines was made at Belle Glade, Florida, during the winter of 1945–46. No protective fungicides were applied. Before the selections were made the plants were scored on the basis of apparent resistance to *Cercospora apii*. A score based on 10 points for complete susceptibility

was used. Lines scoring 1 or 2 had less blight than usually occurs in well-sprayed fields of commercial varieties. Scores above 3 would indicate too much blight, and if above 6 the celery would be worthless. On this basis three lines were scored 1; five were scored 2; ten were scored 3; twelve were scored 4; two were scored 5; two were scored 6; and one was scored 7. The Kilgore's Pride variety grown in the same plots was scored 8. Figure 1 shows a blight resistant F_3 line at the left, and a susceptible commercial variety at the right.

The resistance to *Cercospora apii* in these new celeries is due to more than a single genetic factor. There has been a segregation of plants intermediate between fully resistant and completely susceptible in the F_2 and F_3 generations. Even in the more resistant lines, there is evidence that the expression of resistance is modified by age and unfavorable cultural conditions. The green plants are usually more resistant than the self-blanching types. Most of the self-blanching segregates proved to be susceptible.

Green color in the petioles is dominant over the self-blanching habit. F_2 ratios approximating 3 green to 1 blanched indicate that the green color is due to a single dominant factor. The intensity of the green color varies and appears to be controlled by modifying factors.

Hollow-petiole, or pithiness, was fully dominant in the F_1 and showed an F_2 segregation approximating a 3 to 1 ratio at Ithaca, while at Belle Glade the percentage of solid-petioled plants in the F_2 seemed to be much lower. This suggests that there may be a modifying factor or factors affecting the expression of the 3 to 1 ratio for pithiness under certain climatic or cultural conditions.

Red color which occurs in the petioles of some lines is inherited as a simple dominant. The F_2 generation showed a segregation of 3 red to 1 non-red. This color is expressed most strongly near the base of the petioles.

The inheritance of the factors for ribbed or smooth petioles has not been studied, although it was observed that the majority of the lines are more or less ribbed. A few of the F_3 selections had extremely smooth petioles.

The quality of several of the F_3 selections is high. They have very heavy, crisp petioles, and are not stringy. In some lines the base of the petiole is somewhat flared, and thickened. Rib length varies from 7 to 15 inches in the different lines which have been selected. The fact that the best selections are of the green sorts should be no hindrance to the development of new commercial varieties from them. Pascal-type celeries have been gaining in popularity and may ultimately supplant the self-blanching celeries in consumer preference. The new celeries have an unusual flavor which is delicious in mixed green salads.—G. R. TOWNSEND, Everglades Experiment Station, Belle Glade, Florida, and R. A. EMERSON, Department of Plant Breeding, and A. G. NEWHALL, Department of Plant Pathology, Cornell University, Ithaca, N. Y.